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THIRD SERIES FOUNDED BY EDWARD JACKSON

CONTENTS

DEPARTMENTS

For a complete table of contents see page xxix

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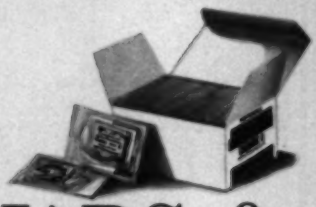

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References:
1. Boland, E. W.: California Med. 88:417 (June) 1958. 2. Bunim, J. J., et al.: Arthr. & Rheum. 1:313 (Aug.) 1958 3. Boland, E. W., and Headley, N. E.: Paper read before the Am. Rheum. Assoc., June 21, 1958, San Francisco, Calif. 4. Bunim, J. J., et al.: Paper read before the Am. Rheum. Assoc., June 21, 1958, San Francisco, Calif.

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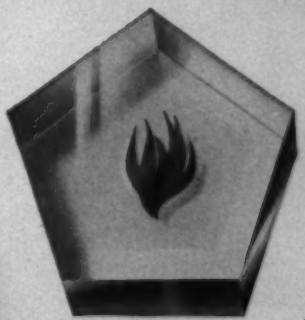

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*Council on Pharmacy and Chemistry: New and Nonofficial Remedies, Philadelphia, J. B. Lippincott Co., 1956, p. 505.

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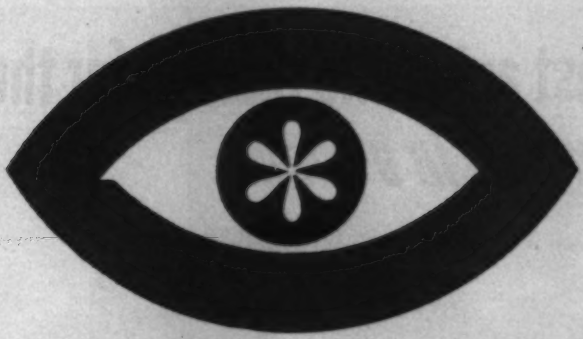

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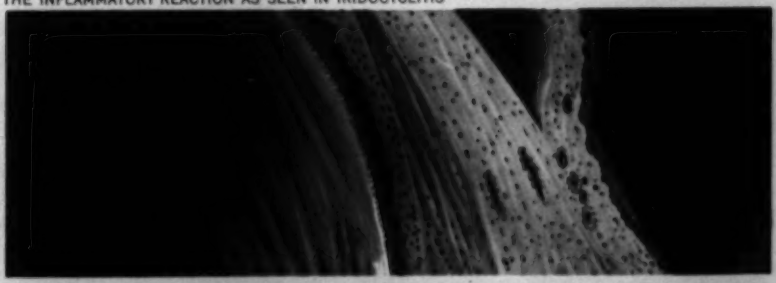
EXUDATIVE INFLAMMATION OF THE UVEAL TRACT

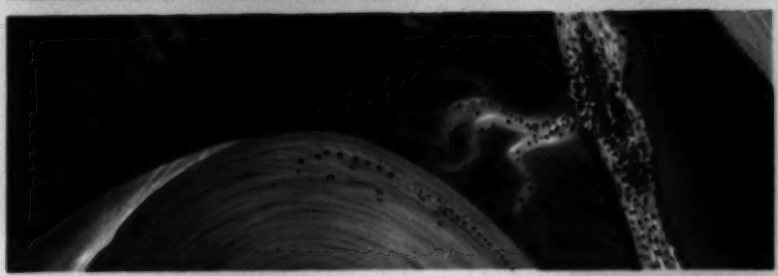
THE INFLAMMATORY REACTION AS SEEN IN IRIDOCYCLITIS

A. Normal tissue. From left to right: posterior surface of ciliary ring, ciliary retina, pigmented layer, ciliary muscle, sclera, and conjunctiva.

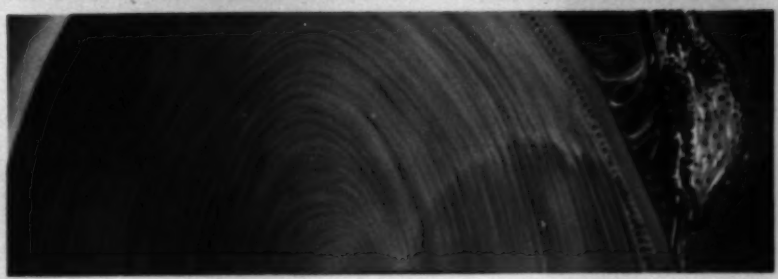


B. Hyperemia is the result of reaction to an endogenous toxin or allergen. Blood vessels are dilated. There is heightened local heat and color. Escape of blood plasma causes edema.

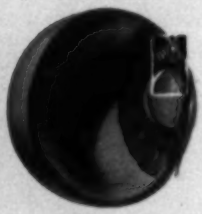
C. The next stage is that of exudation, shown here as macrophages and fibrin streaming from the posterior surface of the iris.



D. As the condition becomes subacute, there is concomitant active exudation and repair. With chronicity, fibroblasts proliferate, new capillaries develop, and various kinds of white cells appear. Formation of a posterior synechia is shown.



SCHWENK

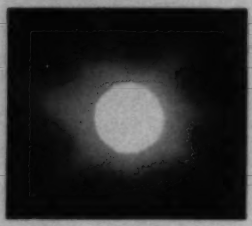


IN OCULAR INFLAMMATORY DISEASES Medrol

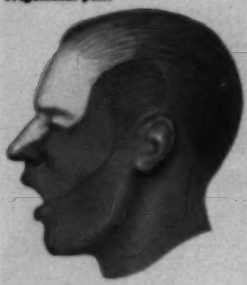
A SYNOPSIS OF OCULAR INFLAMMATORY DISEASE (IRIDOCYCLITIS)

Subjective symptoms:

Photophobia



Trigeminal pain

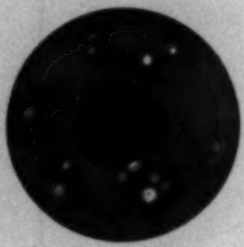


Blurred vision

Lacrimation

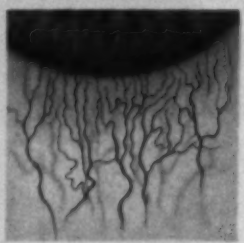
Clinical findings:

Hazy iris



Swollen upper lid

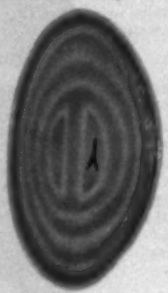
Ciliary injection



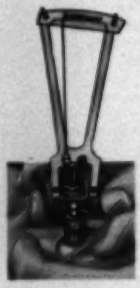
Sequelae: (if untreated)

Anterior and posterior synechiae

Lenticular changes



Secondary glaucoma



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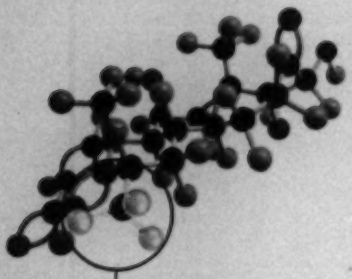

1. Feinberg, S. F.: Medrol in Allergic Conditions: Clinical and Experimental Findings, Metabolism 7:477 (July) 1958.
2. Gordon, D. M.: Methylprednisolone in Ophthalmology, Metabolism 7:569 (July) 1958.

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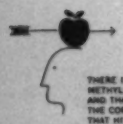

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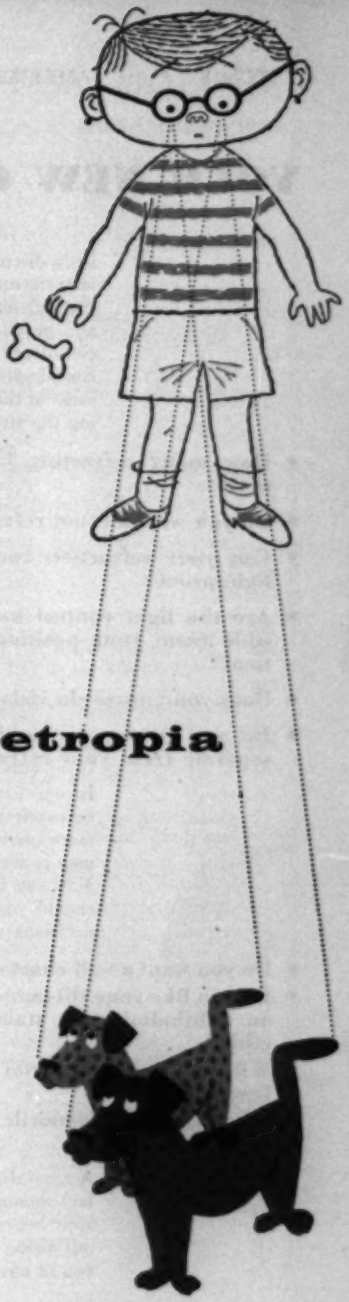

Antimetropia

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CONTENTS

AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 46 DECEMBER, 1958 NUMBER 6

PARALYSIS OF THE THIRD, FOURTH AND SIXTH CRANIAL NERVES*

C. WILBUR RUCKER, M.D.
Rochester, Minnesota

A study of 1,000 cases of paralysis of the nerves to the extraocular muscles was undertaken in order to determine the relative frequency of the various causes. Information on the subject is scanty, and what there is points to a changing incidence of some of the etiologic agents and clearer recognition of others. It is hoped that specific information regarding their relative importance will aid the diagnostician in his selection of tests. It may aid him, too, in the management of those cases in which he fails to find the cause; at least it may offer him something to watch for while he has the patient under observation. Furthermore, historians of future years may find some interest in a record, such as this, of the causes of ocular paralysis as they appeared to the groping diagnostician during the middle decades of the twentieth century.

This is a continuation of a review begun several years ago and reported briefly and in part in THE JOURNAL[1] in 1956. Since then enough more cases have been added to bring the total to 1,000. As noted in the preliminary report, this study was restricted to paralysis of the third, fourth, and sixth cranial nerves, and discarded from it were cases of supranuclear lesions, congenital anomalies, disorders of the muscles themselves, such as myasthenia gravis and ophthalmopathy of Graves' disease, and birth injuries. The latter were excluded because of their frequent complexity and the difficulty in learning exactly what had happened.

* From the Section of Ophthalmology, Mayo Clinic and Mayo Foundation. The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

RESULTS

The 1,000 cases were divided into six groups according to the nerve or combination of nerves in which paralysis was present (table 1). Paralysis of the third cranial nerve accounted for about a third of the cases, paralysis of the sixth cranial nerve for somewhat more than another third. Paralysis of the fourth nerve was relatively infrequent, as were the various combinations. No example was encountered of paralysis of the fourth and sixth nerves together, with sparing of the third.

THIRD CRANIAL NERVE

In 95 of the 335 cases of paralysis limited to the third cranial nerve (table 2), the cause was not determined during the time the patient was under observation at the Mayo Clinic. In most instances the patient was seen only once, but as a rule comprehensive general and neurologic examinations were conducted on that occasion.

Injury to the head accounted for paralysis in 51 cases, in 22 of which injuries were sustained in automobile accidents. Neoplasm was uncommon in this group and accounted

TABLE 1

ACQUIRED OCULAR PARALYSIS: CRANIAL NERVE AFFECTED

Nerve Affected	Cases
3	335
4	67
6	409
3, 4	53
3, 6	76
3, 4, 6	60
Total	1,000

TABLE 2

CAUSES OF PARALYSIS OF CRANIAL NERVE 3

Cause	Cases
Undetermined	95
Head trauma	51
Neoplasm	35
Vascular disease	63
Aneurysm	64
Other	27
Total	335

for paralysis in only about 10 percent of the cases. Twenty-two of the 35 neoplasms arose within the cranial cavity; 13 were metastatic.

The paralysis in 63 instances was attributed to vascular disease, on the basis of evidence of hypertension or arteriosclerosis. As all diagnoses were made on clinical evidence and the pathogenesis of the paralysis was presumptive, many of the cases might have been classified more accurately as undetermined. Diabetes was present in 21 of the 63 vascular cases, apparently contributing to the diseased condition of the vessels.

Aneurysm of the circle of Willis was the most frequent cause of third-nerve paralysis, accounting for 64 of the 335 cases (about 20 percent), nearly twice as many as were caused by neoplasm.

In the other 27 cases in this group paralysis was attributed to a wide range of affections, of which syphilis accounted for six; encephalitis, three; herpes zoster ophthalmicus, three; migraine, two; and poliomyelitis, measles, encephalitis, sarcoidosis, periarteritis nodosa, temporal arteritis, frontal lobe abscess, lupus erythematosus, neuronitis, multiple sclerosis, alcohol injection for trigeminal neuralgia, surgery for brain tumor, and sequela of a myelogram, one each.

The pupillary reactions in the cases in which the third nerve was affected merit comment (table 3). When paralysis was due to tumor or aneurysm the pupils usually exhibited dilatation or incomplete reactions or both. This was true in 28 of the 35 cases of tumor, and 62 of the 64 cases of aneurysm. In the two cases of aneurysm in which the

TABLE 3

CONDITION OF PUPILS IN PARALYSIS OF THIRD CRANIAL NERVE

Cause of Paralysis	Pupils	
	Normal	Affected
Neoplasm	7	28
Aneurysm	2	62
Syphilis	0	6
Vascular disease	52	11

pupillary reactions were normal the diagnosis of aneurysm had been based on clinical evidence alone and arteriography had not been done. The diagnosis was presumptive. In all six cases of paralysis due to syphilis the pupils were affected in one way or another. On the other hand, in the cases attributed to a vascular disorder, pupillary reactions were usually normal. The pupils exhibited abnormalities in only 11 of the 63 cases in which paralysis was ascribed to vascular disease. Within this group, only three of the 21 having diabetes showed abnormal reactions or dilatation. Within the other groups the condition of the pupils was less impressive.

FOURTH CRANIAL NERVE

In only nine of the 67 cases of paralysis of the fourth cranial nerve the cause was undetermined (table 4). Head injuries accounted for 24 cases, 10 of which were incurred in automobile accidents. In only three cases was paralysis associated with malignant disease: in two, metastatic lesions developed from primary lesions in distant portions of the body, and in one an epidermoid cyst which had arisen near the gasserian

TABLE 4

CAUSES OF PARALYSIS OF CRANIAL NERVE 4

Cause	Cases
Undetermined	9
Head trauma	24
Neoplasm	3
Vascular disease	24
Aneurysm	0
Other	7
Total	67

ganglion recurred five years after removal. Vascular disease accounted for the largest number, 24 cases. In most of these one could only speculate as to whether there had been an occlusion of a nutrient artery to the nerve, hemorrhage into the nerve, or direct pressure by the neighboring posterior cerebral or superior cerebellar artery. Not a single case of isolated paralysis of the superior oblique was attributed to aneurysm. Of the miscellaneous group, four were associated with encephalitis and one with neuronitis, while one followed neurolysis of the trigeminal root and one followed ligation of the internal carotid artery for aneurysm.

Sixth cranial nerve

In about a third of the cases of paralysis of the sixth cranial nerve (table 5) the cause was not determined even after thorough examinations, although it is true that many of the patients were seen only once and follow-up studies were not conducted. Head injury accounted for paralysis in 57 cases, with automobile accidents responsible in 30.

Brain tumors were held responsible for sixth-nerve paralysis in 82 cases. This number does not represent all cases that were encountered during the 13 years covered by this survey, for abducens paralysis was regarded as such a minor complication in a patient who had a brain tumor that the diagnosis frequently was not recorded and an unknown number of cases were overlooked in gathering this material. As somewhat similar circumstances existed in some of the other paralyses, the relative error of this factor may thereby be somewhat minimized.

TABLE 5

Causes of paralysis of cranial nerve 6

Cause	Cases
Undetermined	129
Head trauma	57
Neoplasm	82
Vascular disease	57
Aneurysm	16
Other	68
Total	409

Thirty-six neoplasms arose within the cranial cavity, 46 outside it. The neoplasms did not necessarily invade the sixth nerve directly. Some neoplasms, by increasing the intracranial pressure, were responsible for displacement of structures within the intracranial cavity and consequent pressure or traction on the sixth nerve. One explanation of this effect is based on the observation that as pressure within the intracranial cavity increases the only large opening through which the contents may escape is the foramen magnum. The cerebellum drops down and its tonsils herniate into this opening. As the brain stem descends, it pulls the abducens nerve taut between its attachments at the lower margin of the pons and at the apex of the petrous pyramid of the temporal bone. Interruption of the nerve may occur at the tip of the petrous pyramid where the nerve bends forward at a right angle over the bony ridge[2] or anywhere along its course, as a result of traction.[3]

Fifty-seven cases were placed in the vascular subgroup on the basis of hypertensive disease or arteriosclerosis. The nature of the involvement of the nerve is speculative. Diabetes was present in 15 cases and may have been a factor contributing to the vascular disease.

Aneurysm of the circle of Willis was responsible for abducens paralysis in 16 cases. Diagnoses were proved by arteriography in only three of these cases; in the rest they were based on clinical evidence alone and were subject to its errors and uncertainties.

Other miscellaneous causes accounted for 68 cases. In 29 of the 68 cases the causes were attributed to various types of inflammation: in 16 to meningitis or encephalitis, in two to poliomyelitis, in four to mastoiditis, and in seven to syphilis. Of the remaining 39 cases, 15 were attributed to multiple sclerosis and 24 to miscellaneous factors which included injection of alcohol for trigeminal neuralgia and carotid-cavernous fistula, three cases each; spinal puncture, operation for

TABLE 6

CAUSES OF PARALYSIS OF CRANIAL NERVES 3 AND 4

Cause	Cases
Undetermined	14
Head trauma	7
Neoplasm	12
Vascular disease	6
Aneurysm	13
Other	1
Total	53

TABLE 8

CAUSES OF PARALYSIS OF CRANIAL NERVES 3, 4 AND 6

Cause	Cases
Undetermined	10
Head trauma	13
Neoplasm	20
Vascular disease	0
Aneurysm	8
Other	9
Total	60

trigeminal neuralgia, hydrocephalus, and pseudotumor cerebri, two cases each; and spinal anesthesia, ligation of internal carotid artery, surgical treatment for pituitary tumor, subarachnoid hemorrhage, subdural hematoma, cerebral hematoma, chronic alcoholism, sinusitis, excessive vomiting, and cyanosis of congenital heart disease, one case each.

MULTIPLE CRANIAL NERVES

The cause of paralysis of the combined third and fourth nerves and of the third and sixth nerves was not determined in about a third of the cases (tables 6 and 7). No single cause dominates the picture. Noteworthy is the small number of cases ascribed to vascular disorders.

The neoplasms involved in paralysis of the combined third and fourth nerves were as follows: three metastatic tumors, five pituitary tumors, one meningioma, one oligodendroglioma, one neurofibroma, and one pinealoma. The cause listed as "other" in the single case was herpes zoster ophthalmicus.

The neoplasms which caused paralysis of the third and sixth nerves were as follows: metastatic tumors in four cases, pituitary tumor in one case, meningiomas in four cases, chordomas in three cases, and glioma in one case. The other miscellaneous causes were carotid-cavernous fistula in three instances, and one example each of herpes zoster ophthalmicus, alcohol injection of the trigeminal nerve, surgical section of the trigeminal nerve, surgical removal of a tumor in the cerebellopontine angle, encephalitis, meningitis, syphilis, and periarteritis nodosa.

TABLE 7

CAUSES OF PARALYSIS OF CRANIAL NERVES 3 AND 6

Cause	Cases
Undetermined	25
Head trauma	16
Neoplasm	13
Vascular disease	3
Aneurysm	8
Other	11
Total	76

When all three nerves were involved (table 8), the disease process was widespread and the damage extensive. The cause was undetermined in relatively fewer cases in this group than in any of the other groups. Of the 13 cases due to trauma seven were the result of automobile accidents. Neoplasms comprised the largest group. Of the 20 tumors, six were meningiomas; three, pituitary tumors; six, metastatic carcinomas, and two, malignant growths in the nasopharynx; one was a neurofibroma, one was a chordoma, and one was not identified. Paralysis was not ascribed to vascular disease in any of these cases. Aneurysms accounted for eight. The other miscellaneous causes included encephalitis and carotid-cavernous fistula in two cases each and sarcoidosis, syphilis, inflammation at the apex of the orbit, ethmoid operation, and section of the trigeminal root in one case each.

COMMENT

All cases of acquired ocular paralysis included in this study are listed in Table 9. The total numbers in the last column indi-

TABLE 9

CAUSES OF PARALYSIS OF CRANIAL NERVES AND NERVES AFFECTED

Cause	Number of Cases for Each Affected Cranial Nerve						Total
	3	4	6	Combined Cranial Nerves			
				3 and 4	3 and 6	3, 4 and 6	
Undetermined	95	9	129	14	25	10	282
Trauma to head	51	24	57	7	16	13	168
Neoplasm	35	3	82	12	13	20	165
Vascular disease	63	24	57	6	3	0	153
Aneurysm	64	0	16	13	8	8	109
Other	27	7	68	1	11	9	123
Total	335	67	409	53	76	60	1,000

cate that the largest group by far comprised those of undetermined origin which accounted for 28 percent of the whole. This is a greater proportion than I have found reported in any comparable group; for example, Sauvineau[4] (1908) reported 17 percent; Bielschowski[5] (1939), 15 percent; Ochaporski[6] (1942), 14 percent, and Sanna[7] (1956), 22 percent. Had a convenient vascular group not been handy the group having undetermined origin would have been even larger than it was. While this does not necessarily indicate that diagnostic ability is less capable at the Mayo Clinic than in other institutions, it does point out an embarrassing deficiency.

The next group, that of trauma to the head, is distinguished chiefly by the relatively large number of cases of paralysis of the fourth nerve that it contains. About half the injuries were incurred in automobile accidents. The mechanism may be fracture through the sphenoid bone, hemorrhage into the sheath of the nerve, or stretching of the nerve in the posterior fossa as the brain stem is displaced at the moment of violence.[8]

Most of the cerebral neoplasms involved the sixth nerve; remarkably few involved the fourth. There was no significant preponderance of any one type of tumor in the group as a whole, but detailed analysis demonstrated that some types of tumor tended to interrupt specific nerves (table 10). When pituitary tumors caused paralysis they always affected the third nerve, most often alone, but sometimes in combination with one of the others. No example was encountered of this tumor affecting the fourth or sixth nerve alone. Meningiomas did not exhibit special characteristics in this regard, and were surprisingly infrequent as a cause of ocular paralysis. The other primary intracranial tumors, and also the metastatic tumors, predominantly involved the sixth nerve. This was especially true in regard to tumors arising in the nasopharynx, all but one of which involved the sixth nerve, alone or in combination with others. The one tumor involved the third and fourth nerves

TABLE 10

INTRACRANIAL NEOPLASM: EFFECT ON CRANIAL NERVES

Type of Neoplasm	Cranial Nerves Affected						Total
	3	4	6	3 and 4	3 and 6	3, 4 and 6	
Pituitary tumor	14	0	0	5	1	3	23
Meningioma	3	0	5	1	4	5	18
Other primary	4	1	31	3	4	4	47
Nasopharyngeal	0	0	20	1	2	3	26
Other metastatic	14	2	26	2	2	5	51
Total	35	3	82	12	13	20	165

together, causing a paratrigeminal syndrome and narrowing of the pupil. In no case did the tumor paralyze an isolated third or fourth nerve. In three cases paralysis of the sixth nerve was bilateral. Most of the tumors were classified by the pathologist as lymphoepitheliomas. Such tumors are thought to invade the cranial cavity through the foramen lacerum; as they meet the gasserian ganglion on their way upward the common early symptoms are pain and numbness in the distribution of the fifth nerve, followed shortly by abducens paresis.[9,10] The other metastatic tumors originated in scattered sites, most frequently the breast.

To turn to Table 9, it can be seen that vascular disease, like trauma to the head, is distinguished by the relatively large number of cases of paralysis of the fourth nerve. Approximately a third (45 cases) of the 153 cases attributed to a vascular disorder occurred in patients who had diabetes. It was assumed that diabetes was responsible for the vascular disease, which in turn affected the nerves. Forty-five cases in 1,000 is approximately the total incidence of diabetes found among patients admitted to the clinic for all causes. The nerve affected and the number of cases in diabetics were as follows: third nerve, 21; fourth nerve, five; sixth nerve, 15; third and fourth nerves, two; third and sixth nerves, two. When the third nerve was involved, the paralysis was seldom complete and often only two or three muscles were affected. Among the 21 cases of isolated paralysis of the third nerve in diabetic patients the pupils were normal in all except four, and, even in these, iridoplegia was incomplete. The pathogenesis in a case examined at necropsy was demonstrated by Dreyfus and associates[11] to be an ischemic infarct within the center of the nerve trunk due to occlusion of a nutrient artery. In this location it could readily spare the pupillary fibers which lie along the superior surface.[12]

Aneurysm paralyzed the third nerve more frequently than it did any of the other cranial nerves. In only two of the 64 cases of paralysis of an isolated third nerve were the pupillary fibers spared, and in neither of these was the clinical diagnosis confirmed by arteriography. These data are consistent with those of most other reports on the subject in establishing the value of pupillary reflexes in differentiating aneurysm from occlusive vascular disease as a cause of third-nerve paralysis. When pain in the eye or the side of the head accompanies oculomotor paralysis, as it sometimes does when diabetes is the cause[13,14] and nearly always does when aneurysm is the cause, the diagnostician may be hard pressed to distinguish one from the other. The pupillary reactions yield valuable information. If they are normal the paralysis is probably due to occlusive vascular disease; if there is iridoplegia, paralysis is more likely due to aneurysm.

The effects of aneurysm on the cranial nerves have been studied at necropsy by Hyland and Barnett[15] and described under the following two main categories: (1) sudden enlargement of the aneurysmal sac which caused half the cases and was manifested through stretching of the nerve, edema, or intraneural hemorrhage from venous obstruction and consequent fibrous proliferation; (2) other mechanisms which accounted for the other half and included midbrain hemorrhage, kinking and displacement of the posterior cerebral artery, compression by herniation of the hippocampal uncus, and stretching or compression of the brain stem through sudden hemorrhage. My material in its present form does not lend itself to localization of an aneurysm on specific vessels in the circle of Willis on the basis of the nerve affected.

Other causes of paralysis of the third, fourth, and sixth cranial nerves comprised a few examples each of a wide variety of disorders. Some of the other causes are listed in Table 11. The infections were largely various forms of encephalitis and meningitis. Four of these followed mastoiditis and led to Gradenigo's syndrome with its abducens paresis.

TABLE 11

MISCELLANEOUS CAUSES OF PARALYSIS OF CRANIAL NERVES

Cause	Cranial Nerve			
	3	4	6	Combinations
Infections	5	5	16	6
Poliomyelitis	1	0	2	0
Syphilis	6	0	7	2
Multiple sclerosis	1	0	15	0

Poliomyelitis accounted for one case of paralysis of the third nerve and two of the sixth, so few as to be without numerical significance. Some cases undoubtedly were overlooked, as I have not reviewed the clinic records of all poliomyelitis patients registered during the period of this survey. Two reported surveys may serve as examples. One covered the years 1950 to 1952 at Baltimore[16] and showed six cases of sixth-nerve paresis, in two of which the third nerve was involved additionally and in another both the third and fourth nerves in addition to the sixth. In the epidemic at Cleveland[17] in 1953 through 1954, the third nerve was said to be involved in three of the 500 cases, but whether fibers to the pupils or eye muscles were affected is not stated; the sixth nerve was interrupted in 10; the fourth was not involved. Clearly, in poliomyelitis abducens paralysis is the most frequent ocular complication.

Among the cases attributed to syphilis (table 11) there was no example of isolated trochlear paralysis. In every case in which the third nerve was involved there was some degree of iridoplegia. The total of only 15 cases of syphilis in 1,000 cases of ocular paralysis collected since 1944 presents a vivid picture of the efficacy of recent therapy. Compare this incidence of 1.5 percent with that in four other studies selected somewhat at random: Sauvineau[4] (1908, France) 48 percent, Bielschowski[5] (1939, Germany) 26 percent but 55 percent in his earlier statistics, Ochaporski[6] (1942, Russia) 52 percent, Sanna[7] (1956, Italy) four percent.

Multiple sclerosis accounted for paralysis in 16 cases: sixth-nerve paralysis in 15 and third-nerve paralysis in one. There was no example of fourth-nerve paralysis, a finding encountered also by others.[18, 19] Those patients who had multiple sclerosis and who gave a history of having recovered from diplopia were not counted in this survey. The single example of what appeared to be partial paresis of the third nerve concerned a patient who presented a slight weakness of the right inferior and superior rectus and inferior oblique muscles and a horizontal nystagmus of small amplitude on gaze to each side. There were a few cases of slight weakness of one or two of the vertically acting muscles, but in each instance there was associated hyperphoria or skew deviation or rotary nystagmus that appeared to account satisfactorily for the apparent weakness. The examples of paresis of one or both medial recti were attributed to internuclear lesions, and therefore discarded from this survey, as were the supranuclear lesions. Disturbances of the pupillary reactions, aside from those due to visual loss, that could be unequivocally ascribed to multiple sclerosis were not encountered in this survey and must be rare.

Other miscellaneous causes occurred in such small numbers that a review of them is pointless. Each is noted under the discussion of the nerve affected.

SUMMARY

A review of 1,000 cases of paralysis of the third, fourth, and sixth cranial nerves disclosed that the cause was not found in 28 percent. Head injuries, about half of which were incurred in automobile accidents, accounted for 17 percent of the cases. An equal number were due to brain tumors, nearly half of which were metastatic. Vascular disease was thought to be responsible for about 15 percent. Aneurysm of the circle of Willis explained the paralysis in 11 percent of the cases, the majority involving the third nerve. The cause of paralysis in the remaining 12 percent was associated with a diversity of disorders.

The Mayo Clinic.

References

1. Rucker, C. W., Dyer, J. A., Smith, D. C., and Taub, R. G.: The causes of acquired paralysis of the ocular muscles. Am. J. Ophth., **41**:951-955 (June) 1956.
2. Wolff, E.: The Anatomy of the Eye and Orbit. (Including the central connections, development, and comparative anatomy of the visual apparatus.) New York, Blakiston, ed. 4, 1954, p. 260.
3. Collier, J.: The false localising signs of intracranial tumour. Brain, **27**:490-508, 1904.
4. Sauvineau, C.: Paralysies des muscles de l'oeil. In Lagrange, F., and Valude, E.: Encyclopédie Francaise D'ophthalmologie. Paris, Octave Doin, 1908, v. 7, pp. 585-846.
5. Bielschowski, A.: Ätiologie der augenmuskellähmungen. In: Graefe-Saemisch Handbuch. Berlin, Verlag Von Julius Springer, 1939, ed. 2, v. 8, pt. 1, pp. 433-457.
6. Ochaporski: Quoted by Scobee, R. G.: The Oculorotary Muscles. St. Louis, Mosby, 1947, p. 193.
7. Sanna, M.: Eziologia e frequenze della paralisi oculari estrinseche. Rev. oto-neuro-oftal., **31**:425-447 (Sept.-Oct.) 1956.
8. Rowbotham, G. F.: Acute Injuries of the Head: Their Diagnosis, Treatment, Complications, and Sequels. Baltimore, Williams & Wilkins, 1942, p. 194.
9. Godtfredsen, Erik: Ophthalmo-neurological symptoms in connection with malignant nasopharyngeal tumours. Brit. J. Ophth., **31**:78-100 (Jan.) 1947.
10. Lawley, M.: Nasopharyngeal carcinoma: An account of the cranial nerve lesions found in 185 cases. Australian & New Zealand J. Surg., **25**:170-178 (Feb.) 1955-1956.
11. Dreyfus, P. M., Hakim, S., and Adams, R. D.: Diabetic ophthalmoplegia. Arch. Neurol. & Psychiat. **77**:337-349 (Jan.) 1957.
12. Sunderland, S., and Hughes, E. S. R.: The pupillo-constrictor pathway and the nerves to the ocular muscles in man. Brain, **69**:301-309, 1946.
13. Weinstein, E. A., and Dolger, Henry: External ocular muscle palsies occurring in diabetes mellitus. Arch. Neurol. & Psychiat., **60**:597-603 (Dec.) 1948.
14. Eareckson, V. O., and Miller, J. M.: Third-nerve palsy with sparing of pupil in diabetes mellitus: A subsequent identical lesion of the opposite eye. Arch. Ophth., **47**:607-610 (May) 1952.
15. Hyland, H. H. and Barnett, H. J. M.: The pathogenesis of cranial nerve palsies associated with intracranial aneurysms. Proc. Roy. Soc. Med., **47**:141-146 (Feb.) 1954.
16. Murray, R. G., and Walsh, F. B.: Ocular abnormalities in poliomyelitis and their pathogenesis. Canad. M. A. J., **70**:141-147 (Feb.) 1954.
17. Engler, C. W., and Missal, S. C.: Involvement of the cranial nerves in poliomyelitis. Tr. Am. Acad. Ophth., **59**:732-739 (Nov.-Dec.) 1955.
18. Savitsky, N., and Rangell, L.: The ocular findings in multiple sclerosis. In: Multiple Sclerosis and the Demyelinating Disease. Baltimore, Williams & Wilkins, 1950, v. 28, pp. 403-413.
19. Marshall, D.: Ocular manifestations of multiple sclerosis and relationship to retrobulbar neuritis. Tr. Am. Ophth. Soc., **48**:487-525, 1950.

IRIDOSCHISIS*

A CLINICAL AND HISTOPATHOLOGIC STUDY

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A seemingly rare disease, reported only 15 times in medical literature, was first described by A. Schmitt[1] in 1922. He called it separation of the anterior layers of the iris. It was Loewenstein and Foster[2] who first gave to this condition the name iridoschisis, which means separation of the iris into two layers and multiple rupture of the iris fibers. It is derived from the Greek work, "schisis," meaning cleavage.

The appearance of this condition is rather characteristic. The anterior layers of the iris

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become separated from the posterior layers. In some areas, the fibers are attached only at the ciliary body and at the iris sphincter and bulge forward in a curve. In some areas, the fibers become torn in two, sometimes at the iris sphincter and sometimes in the middle of the fibers. The fibers thus become separated and the ends float forward in the aqueous like water lilies in a pond. In other areas, only a few fibers seem to be pulled out of the stroma, just as one would tease a few threads out of a piece of cloth with a needle or crochet hook. These loose, floating fibers usually contain a small arteriole and may be straight, slightly curved, or curled. This condition usually appears in the lower half of the iris and, in a typical case, the upper half is grossly normal. The posterior layer of the iris is usually intact and the sphincter and dilator fibers function in the normal manner.

The majority of the cases reported appeared in people over the age of 65 years. In this group, iris atrophy was a common condition and glaucoma was often associated with it. Secondary glaucoma was not a common finding, nor did trauma seem to play a major part in a typical case. Severe lens sclerosis was also a common finding. Changes in the posterior segment of the eye were no more common than in patients in this age group without the disease.

The cause of the disease is unknown. Because the condition appears in the lower half of the iris only, gravity could be a major cause. The weight of the structure of the inner eye on the atrophic fibers, conceivably, might rupture them. Atrophy of the connective tissue of the iris commonly seen as an aging process is probably the major cause. The fibers are thus weakened and some of them rupture and then float forward in the anterior chamber. Because of the peculiar structure of the blood vessels in the fibers, blood has never been seen in the anterior chamber.

Up to the present time, only 15 cases were found reported in medical literature. Schmitt[1] reported a case of separation of the iris in 1922. His report was followed by those of Drabkin[2] (1923), Sander[3] (1925), Vogt[4] (1926), Imre[5] (1927), Dollfus[6] (1927), Loewenstein and Foster[7] (1945), Loewenstein, Foster, and Sledge[8] (1948), and Gardner and Wier[9] (1949). The case reports by Shoenberg[10] (1927), Linn and Linn[11] (1949), Viers[12] (1949), McCulloch[13] (1950), and Haik, Lyda, and Waugh[14] (1952), were the only ones in the American literature. The only case studied histopathologically is that of Loewenstein and Foster.[7] Our case, therefore, is the second one put on record in which a careful histopathologic study of the eyes has been done.

Some of the associated lesions commonly found by the authors just listed were evidences of iritis, iridocyclitis, glaucoma, endothelial corneal dystrophy, arcus senilis, and choroidal rupture. Trauma was thought to have been an etiologic factor in a few cases. General findings were not contributory. It was noted by some that most of the associated findings were those commonly seen in the aging process of the various intraocular structures. One might, therefore, believe that it may be a form of senile atrophy of the iris, but Loewenstein and Foster felt that it was a distinct entity set apart from mere senile degeneration.

Report of a Case

Mrs. J. E. S., aged 85 years, first appeared at the Christie Clinic in September, 1952, complaining of gradual, painless visual failure in both eyes over the past two years. She had had an eye examination 11 years previously by an ophthalmologist now deceased, who gave her a pair of glasses but did not mention that there was anything wrong with her irises. He did tell her that she had mild cataracts.

The patient stated that her general health was excellent, considering her age of 85 years. She reported that she had developed carcinoma of the left breast five years previously, for which she had had a radical mastectomy. Following this, she developed what physicians have considered to be a severe herpes zoster over the left hemicranium, left neck, shoulder, and upper chest region. This was followed by marked residual neuritis, which continued to produce hyperesthesia, grade 4. Review of all other systems was negative. The last chest X-ray film for follow-up of the breast carcinoma was negative in 1952.

Fig. 1 (Albers and Klien). Appearance of the right and left eyes of the case reported. Note the symmetry of the lesions.

The physical examination revealed a scar on the bridge of the nose where a basal-cell epithelioma had been removed by the Dermatology Department. There was moderate superficial scarring of the skin over the left side of the neck, left shoulder, and pectoral region. Throughout the area, hyperesthesia was rather marked. She had an old mastectomy scar on the left chest and axilla. The blood pressure was 140/90 mm. Hg. The heart sounds were normal and the heart was not enlarged. Her lungs were clear. The abdomen was negative.

Examination of the right eye (fig. 1) revealed a vision of counting fingers only. The lids and conjunctiva appeared normal. The cornea suffered from severe arcus senilis and mild endothelial dystrophy. The anterior chamber was slightly shallow. The pupil was round, and regular, and reacted normally. The upper half of the iris was normal. From the 3- to 7-o'clock positions, the iris stroma appeared to be separated from the pigment layer; most of the radial fibers of the iris seemed to be detached at the pupillary margin or broken in two in the middle and were floating freely in the aqueous. Some appeared straight and were single, others appeared in groups of three or four and were slightly arcuate. Other single fibers had curled ends. Each fiber seemed to contain a blood vessel full of bright red blood but there was no free blood in the anterior chamber. There was a little pigment floating in the aqueous but no cells. In several areas where the disease occurred, there were pieces of stroma pasted on the pigment layer. The sphincter of the pupil was intact, but there were a few rarefied areas in the pigment layer. The collarette was absent in the diseased area but was otherwise intact. The lens was uniformly opaque. After the lens was removed, mild choroidal atrophy throughout the fundus and mild arteriolar and choroidal vascular sclerosis were noted. The tension was 18 mm. Hg (Schiøtz).

The vision in the left eye was 20/200 and the patient could read 14/89. This eye (fig. 1), too, revealed severe arcus senilis, mild endothelial dystrophy, a slightly shallow anterior chamber, and a dense nuclear cataract. The iris was normal except from the 5- to 7-o'clock positions where the appearance of the diseased iris was exactly the same as that of its fellow. The tension was 18 mm. Hg (Schiøtz).

Studies with the Thorpe gonioprism revealed that the angle was open. A little more pigment than normal was present over Schlemm's canal. From about the 5- to 7-o'clock positions there were a few clumps of fibers bunched up in the angle so that it could not be seen in that area.

In September, 1952, a successful intracapsular lens extraction was done on the right eye. At that time, a broad iridectomy was done and the excised iris was sent to Dr. Bertha Klien for study. Convalescence was uneventful and the final corrected vision was 20/25 and 14/14.

This patient was seen intermittently after that time. Her irises always appeared the same and the tension remained normal. She developed cardiac decompensation and died in January, 1957. Both eyes were removed by her family physician and were sent to Dr. Klien for study.

Histologic findings

Five years prior to the post-mortem enucleation of both eyes, a piece of the right superior iris was obtained through total iridectomy during the procedure for lens extraction. The piece of iris was embedded in paraffin and the following observations were made in the sections:

The sphincter portion of the iris was nor-

mal. Within the middle portion, the anterior and posterior layers of the iris were separated by a cleft at whose edge arose a few free floating strands of iris stroma, one of them containing a thick-walled blood vessel.

There was no evidence of inflammation. The iridoschisis, which was clinically visible in the lower half of this iris, was merely indicated in the excised upper portion by a line of cleavage, formed probably by senile atrophy of the intermediate tissue which represents the link between the denser anterior and posterior layers of the iris.

The globes obtained post mortem were fixed in formalin, embedded in celloidin, and sectioned serially in the vertical plane.

The iris

In the right eye which had undergone the lens extraction, a stump of iris adhered to the posterior surface of the cornea between the superior chamber angle and the inner end of the postoperative scar. Superior iris, still preserved at each side of the total coloboma in this eye, and the superior half of the left iris were better preserved than the inferior halves, although a distinct line of cleavage separated everywhere the anterior mesodermal from the posterior neuroectodermal portions (fig. 2).

The inferior halves of both irises presented similar pictures. The stroma of the pupillary portion was almost completely absent leaving sphincter and dilator muscles exposed (fig. 3). Loosely connected strands of stroma, some of them carrying thick-walled, blood-filled vessels, were floating in the anterior chambers (figs. 4 and 5).

Within the ciliary portion, shreds of stroma were preserved around strands and clusters of vessels, many of them with homogeneous walls. In some of them, this lack of structure of the walls was due to hyalinization, as shown by the van Gieson stain; in

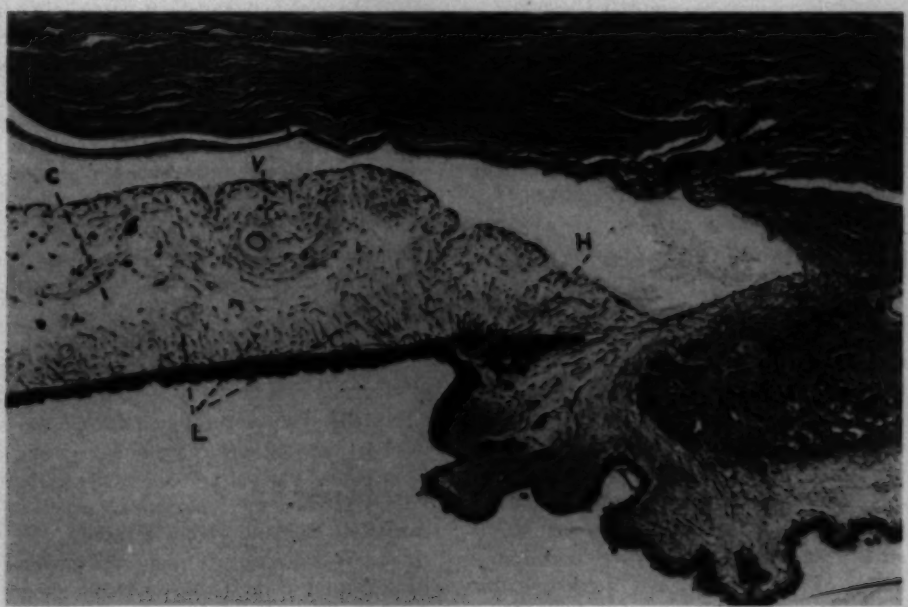

Fig. 2 (Albers and Klien). Inferior ciliary portion of left iris. (C) Line of cleavage formed by atrophy of intermediate tissue. (L) Obliterated vascular lumina near posterior neuro-ectodermal layers of iris. (V) Intact but hyalinized walls of arteriole in anterior portion of stroma. (H) Hamartomalike mass of neuro-ectodermal cells at ciliary ending of dilator muscle. (van Gieson, ×65.)

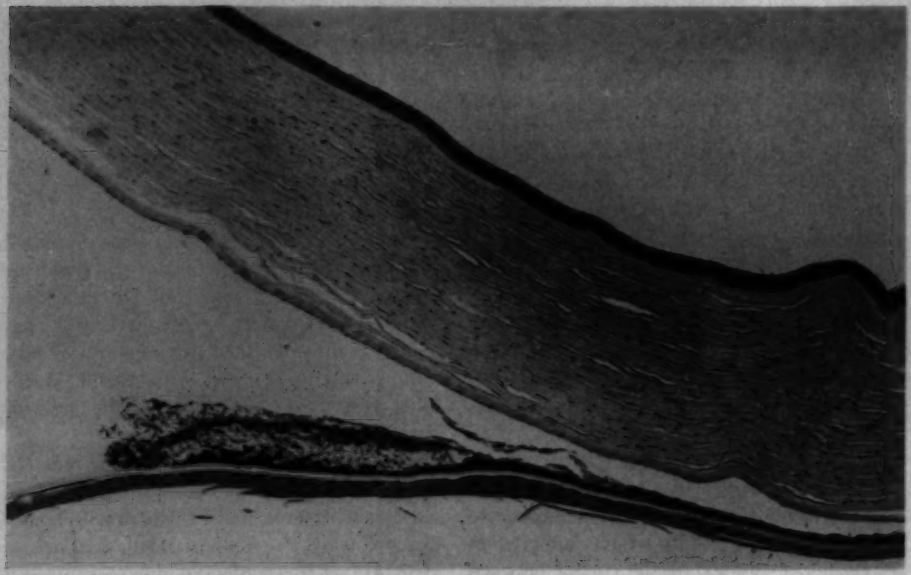

Fig. 3 (Albers and Klien). Inferior pupillary portion of left iris. Only the posterior neuro-ectodermal portion of the iris remained. (Hematoxylin-eosin, ×65.)

others, there merely was a disintegration of the wall which left behind a shadowy outline of the former vascular boundaries (fig. 2). In a number of capillaries and veins, the endothelial cells were laden with pigment, and there was some pigment dispersion in both eyes over the posterior corneal and the trabecular surfaces. Fine pigment granules were also contained within the spaces of Fontana and within the endothelial cells of the collector channels beyond the canal of Schlemm. There was moderate degeneration of the pigment epithelium of the iris, whose cells were flatter than normal, although its continuity was well preserved. At the pupillary border only, dissolution of some cells had led to the pigment dispersion.

The lower angle of both anterior chambers was open; only one small fragment of iris stroma was found lying on the inferior trabecula of the operated eye, suggesting that the apparently free-floating strands elsewhere were still attached to the iris at one point, probably by a central vascular core.

In the inferior half of the right iris, there was a circumscribed thickening of the dilator muscle from which radiated a fanlike structure of spindle cells with interspersed clump cells, very similar to that described by Loewenstein and Foster in their case of iridoschisis. Similar cell accumulations were lying in several places at the ciliary ending of the dilator muscle in both eyes of our patient.

There were no signs of inflammation in either iris.

Remaining structures of the anterior and posterior segments

The cells of the corneal endothelium were irregularly spaced and considerably rarefied in the central portions. At the upper limbus of the right eye, the postoperative scar was remarkably well and evenly healed.

The left lens was in situ and the globular disintegration in the remnants of the otherwise fallen-out cortex indicated a cataract.

There was marked hyalinization of the ciliary processes. The cells of the nonpig-

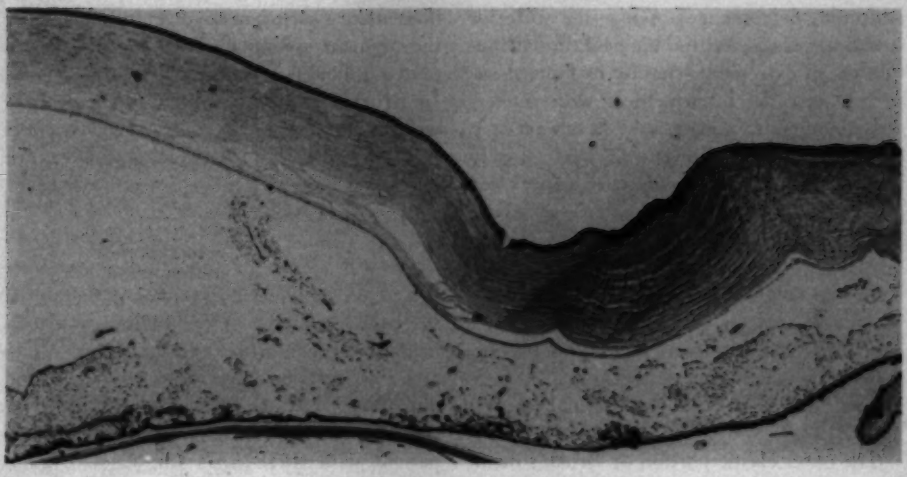

Fig. 4 (Albers and Klien). Strands of left iris stroma, some containing blood-carrying blood vessels, floating in anterior chamber almost touching the cornea. (Hematoxylin-eosin, ×28.)

mented ciliary epithelium were flat and degenerated and, over extensive portions of the orbiculus region, bleblike detachments from the pigmented epithelium had formed. It was noteworthy that the major circle of the iris did not show any sclerotic thickening.

The lower vitreous in both eyes contained a small amount of erythrocytes. This finding, together with the slight edema of the otherwise normal optic nerves and a low peripapillary retinal detachment by transudate was interpreted as due to ante-mortem terminal vascular paralysis.

The retina of the right eye was normal aside from some cystic degeneration at the ora. The left retina contained a few small

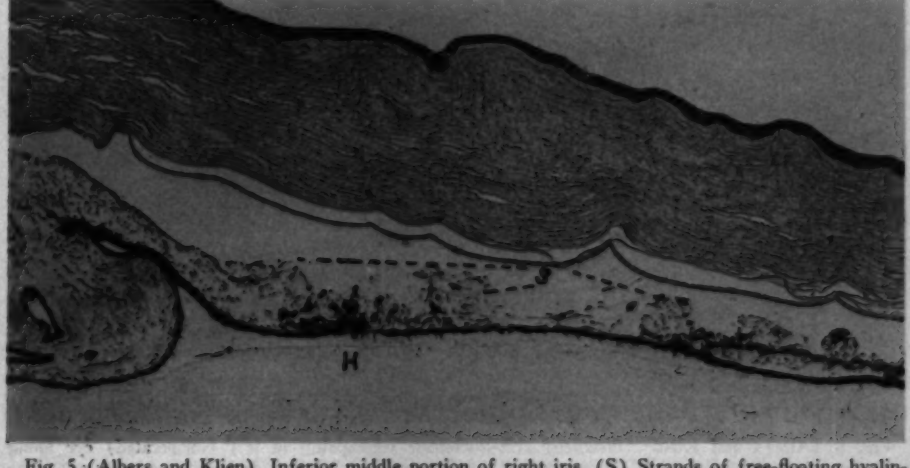

Fig. 5 (Albers and Klien). Inferior middle portion of right iris. (S) Strands of free-floating hyalinized vessels, some still carrying blood. (H) Hamartomalike mass of neuro-ectodermal cells connected with dilator muscle, similar to those in Figure 1. (Hematoxylin-eosin, ×42.)

scattered hemorrhages along the inferior temporal vessels. Within the field of distribution of an extremely sclerotic hyalinized superior arteriolar branch, there were numerous albuminous exudates, and at one place in the outer avascular layers of the retina, a striking new formation of blood vessels had arisen (fig. 6).

The large and medium-sized choroidal arteries had thickened fibrous walls in both eyes, but only in the left eye were there areas of atrophy of the choriocapillaris. Corresponding to the largest of these areas just below the foveal region, there was a circumscribed defect in the first retinal neuron (fig. 7).

The outer elastic portion of Bruch's membrane contained spotty calcifications at short intervals over the entire posterior polar regions, while the inner cuticular portion was thickened by partly granular, partly calcified (silver nitrate stain after Kossa) confluent drusenlike excrescences. Over these plaques the pigment epithelium was flat and degenerated although its continuity was preserved. In Bruch's membrane of the left eye, there were a number of breaks and dehiscences through which fibroblasts had started to proliferate.

Comment

Careful search of the literature has revealed no previous histopathologic study of the pure condition of iridoschisis unassociated with other ocular disease. Loewenstein and Foster,[7] who have written the first and apparently only previous histologic description of this condition, excised one eye of their patient with iridoschisis for absolute glaucoma, and could not rule out completely the effect upon the iris of abnormal (lytic) metabolic products from the long-standing glaucoma.

In our patient, aging processes in other

Fig. 6 (Albers and Klien). Localized arteriosclerotic retinopathy in left (unoperated) eye along sclerotic superior arteriole. (T) Transudate. (H) Hemorrhage. Neovascularization of outer avascular retinal layers. Choriocapillaris well preserved, spotty calcification of Bruch's membrane. (Hematoxylin-eosin, ×145.)

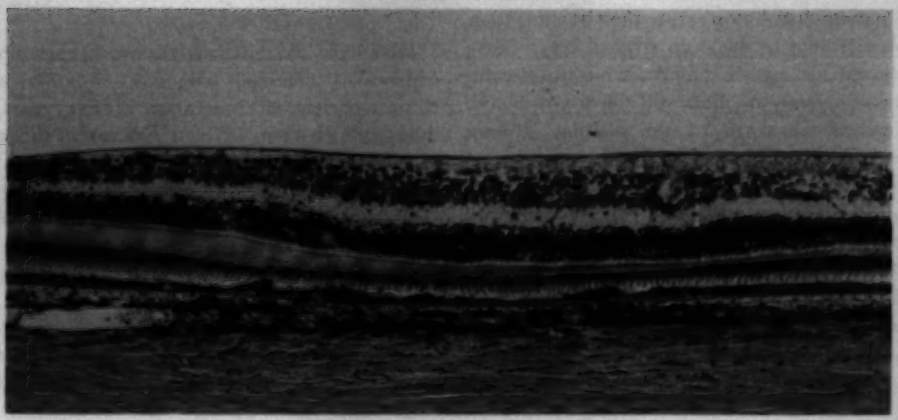

Fig. 7 (Albers and Klien). Localized disturbance of neuro- and pigment epithelium below foveal region in left (unoperated) eye, corresponding to area of circumscribed rarefication of choriocapillaris and larger vessels. (Hematoxylin-eosin, ×135.)

tissues of the anterior and posterior segment were very obvious, some of them being identical in the two eyes. Noteworthy differences were the numerous cracks and dehiscences in Bruch's membrane in the operated eye, and the absence in this eye of the spotty degeneration of the neuro-epithelium corresponding to areas of atrophy in the choriocapillaris, and of the arteriosclerotic retinopathy due to patchy obliterative retinal arteriolar sclerosis, so obvious in the unoperated eye.

One might speculate that the extreme lowering of the intraocular pressure and the manipulations during the lens extraction had damaged the fragile calcified lamina basalis of the choroid, and further consider as a more remote possibility that these events might have improved the circulation in retina and choroid during and for some time after the surgical procedure, postponing or ameliorating the consequences of angiosclerosis.

Regarding the peculiar circumscribed thickening of the dilator muscle found in Loewenstein and Foster's and in our case, it would seem that one is dealing with a developmental anomaly, unrelated to the process of iridoschisis, comparable to the hamartoma-like formations at the ciliary ending of the dilator muscle (Klien[15]). These cell accumulations which show variable mixtures of spindle cells, cylindric cells, and clump cells, in keeping with the pluripotential properties of the cells of the secondary optic vesicle from which they arise, are not uncommon at the ciliary ending of the dilator muscle, appearing to be rarer or perhaps only less noticeable in its middle portions. They were conspicuous in the two cases of iridoschisis because of the disappearance of the surrounding stroma.

The process of iridoschisis appears to begin in the intermediary tissue between the denser anterior stromal and posterior muscular portions, producing a line of cleavage at a rather early stage. Since the play of the normal iris muscles is considerable and continuous and the anterior stroma is criss-crossed by increasingly sclerotic vessels which cannot follow the muscular action with their former elasticity and freedom of motility, the remaining bridges of the intermediary tissue are gradually torn and the anterior and posterior layers of the iris become separated.

The immediate cause of the degeneration of the intermediary tissue is not clear. It is perhaps significant that the vessels

showing dissolution rather than hyalinization were found in the deep stromal layers near the dilator muscle (fig.2), while the hyalin vessels, many of them still filled with blood, formed the cores of the floating stromal strands. Thus, the essential pathologic change may not be sclerosis and hyalinization but an obliterative process. Its basis might be a hemodynamic regulating mechanism by which branches that arise at a more unfavorable angle from the main vascular trunks in a given organ are shut off when circulation is becoming impaired in advanced old age.

Summary and Conclusions

Histologic examination of two eyes with iridoschisis obtained post mortem after a clinical period of observation of five years during which a lens extraction in one of them restored vision to 20/25, permitted the definite statement that neither trauma nor any type of associated ocular disease is the cause of this striking condition. It is in a small way a replica of natural death from the aging process.

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References

1. Schmitt, A.: Ablosung des vorderen Irisblattes. Klin. Monatsbl. f. Augenh., **58**:214-215, 1922.
2. Drabkin, B.: Spontane Loslösung der vorderen mesodermalen Irisplatte. Klin. Monatsbl. f. Augenh., **71**:188-190, 1923.
3. Sander, E.: Erworbene Ablosung des vorderen Irisblattes. Klin. Monatsbl. f. Augenh., **74**:729-730, 1925.
4. Vogt, A.: Abgelöste vordere Irisplatte als Senile Veränderung (Senile Irisschürze). **77**:710, 1926.
5. Imre: Quoted by Shoenberg.*
6. Dollfus, A.: Degénérescence irenni d'un type particulier. Bull. Soc. ophtal. Paris., 1927, p. 170.
7. Loewenstein, A., and Foster, J.: Iridoschisis with multiple rupture of the stromal threads. Brit. J. Ophth., **29**:277-282, 1945.
8. Loewenstein, A., Foster, J., and Sledge, S. K.: A further case of iridoschisis. Brit. J. Ophth., **32**:129-134, 1948.
9. Gardner, R. R., and Wier, A. R.: Iridoschisis in a case of chronic primary glaucoma. Brit. J. Ophth., **33**:509-511, 1949.
10. Shoenberg, J. J.: Case of detachment of the anterior layers of the iris. Arch. Ophth., **56**:538-542, 1927.
11. Linn, J. G., and Linn, J. G., Jr.: Detachment of the anterior layers of the iris. (iridoschisis): Report of a case. Am. J. Ophth., **32**:1700-1702, 1949.
12. Viers, R. R.: Iridoschisis (a case report). Am. J. Ophth., **32**:261-262, 1949.
13. McCulloch, C.: Iridoschisis as a cause of glaucoma. Am. J. Ophth., **33**:1398-1400, 1950.
14. Haik, G. M., Lyda, W., and Waugh, R. L.: Iridoschisis. Arch. Ophth., **48**:40-43, 1952.
15. Klien, B.: The ciliary margin of the dilator muscle of the pupil. Arch. Ophth. **15**:985-993, 1936.

STUDIES ON THE MORPHOLOGY AND PATHOLOGY*

Of the trabecular meshwork in the human eye

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Gonioscopy and the etiology of glaucoma have equally kept awake the interest in the trabecular meshwork. Although the older authors preferred sagittal sections, more recent researchers (Ashton, Brini, and Smith, as well as Busacca, Flocks, Kurus, Rohen, Unger, Vrabec) have also used flat sections or flat preparations in order to obtain a thorough knowledge of the structure of the chamber angle. In our opinion the results thus far obtained may be summed up to show that there are three different tissue formations in the meshwork integrated into a functional unit:

* From the Institute of Anatomy, University of Mainz, Director: Prof. Dr. A. Dabelow; and from the Department of Ophthalmology, Freiburg im Breisgau, Director: Prof. Dr. W. Wegner.

1. An equatorial, elastic reticulum which represents the mechanical basis of the trabeculae and is able to be expanded, indirectly (Unger, and Rohen, 1957) as well as directly, by contraction of the ciliary muscle-choroid system since a large portion of the anterior ciliary muscle sinews join not only the cornea and the scleral spur, but also the elastic reticulum of the meshwork proper (Rohen, 1956).

2. A system of endothelial cells which communicates with the corneal endothelium, the endothelium of the iris, and the endothelium of Schlemm's canal.

3. Light-microscopically homogeneous substances enveloping the elastic fibers and forming flat lamellas (fig. 1).

According to Graumann and Rohen (in press) the following details are to be distinguished: (a) a central ground substance lamella, and (b) a superficial subendothelial basement membrane (glass membrane) which communicates with another basement membrane situated between Descemet's membrane and the corneal endothelium and which has been observed by these authors. Descemet's membrane does not extend to the trabecular meshwork.

The pertinent literature was thoroughly reviewed by Ashton, et al. We have recently published and discussed new results concerning the anatomic and functional structure of the trabecular meshwork (Rohen, 1956, 1957; Unger 1956, 1957) which makes a repetition of our findings in the present study unnecessary.

Materials and Methods

We examined histologically, with regard to alterations of the corneoscleral trabeculae, the chamber angles of enucleated globes with primary or secondary glaucoma and of eyes without increased intraocular pressure, which had to be enucleated because of tumors or perforating injuries. We used sagittal and flat sections. Stains were hematoxylin-eosin, Azan, van Gieson, Masson-Goldner's trichrome stain, PAS-McManus, toluidine blue, and chrome-hematoxylin-phloxin according to Gömöri.

Materials were 14 globes without glaucoma (five from children from two to six years of age); six eyeballs with absolute primary glaucoma from persons 20 to 75 years of age; 22 globes with absolute secondary glaucoma, mostly from persons 30 to 83 years of age; making a total of 42 eyes.

Findings

1. *Two sets of tissue* in the region of the

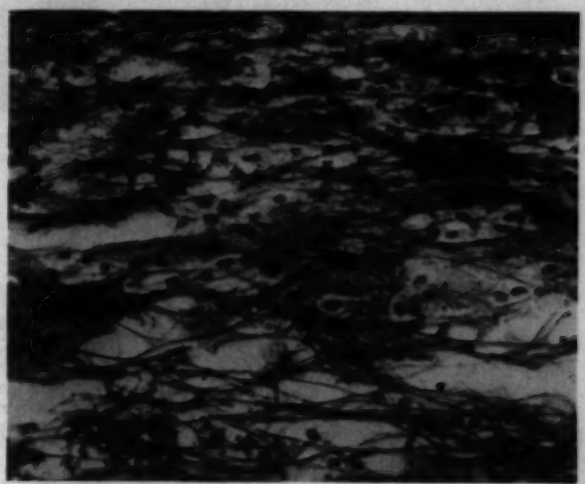

Fig. 1 (Rohen and Unger). Tangential frozen section of the trabecular meshwork. Note the elastic reticula and the endothelial cells spread flatly between the oval pores. (×320.)

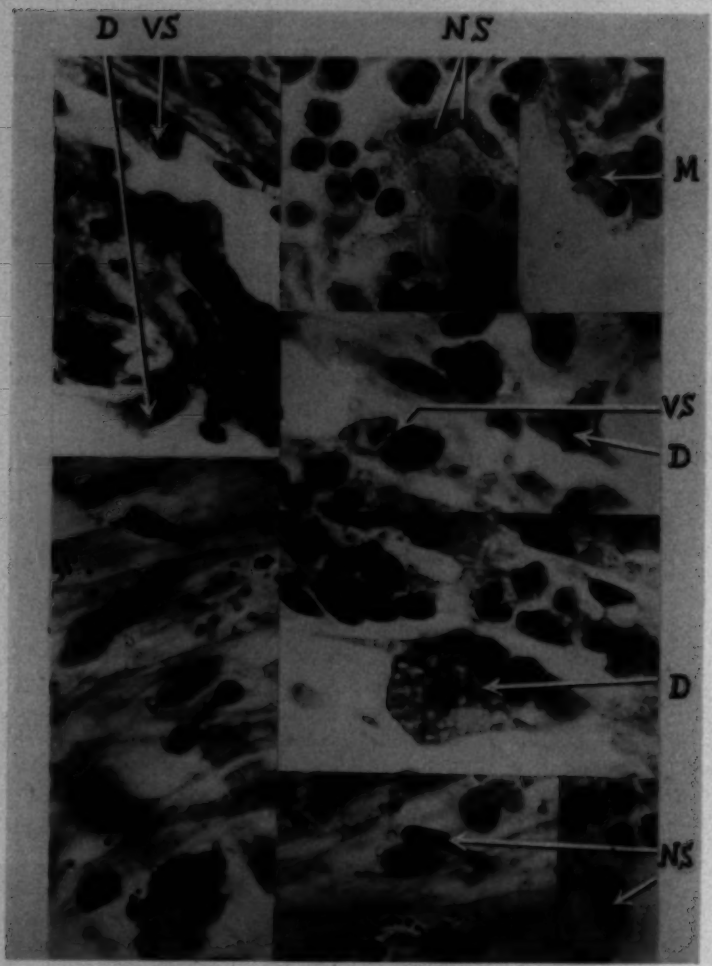

Fig. 2 (Rohen and Unger). Reactions of the trabecular endothelium. Cell desquamations (D), vital storage (VS), mitoses (M), and amitoses as well as numerous different nuclear shapes (NS) are visible. (×1,000.)

trabecular meshwork are of special importance to the aqueous circulation, namely, endothelium and basement membrane.

A. Endothelium of the trabecular meshwork

In the normal eye the endothelium is fixed flatly within the trabecular meshwork (fig. 1). With larger meshes in the reticulum it sometimes happens that pores stay open (Ashton). The smaller ones, however, are as a rule covered completely by endothelium. Mitoses occur, especially in pathologic reactions of the eye (inflammations, glaucoma), and so do, evidently, amitoses, which is disclosed by the curious shapes of the nuclei

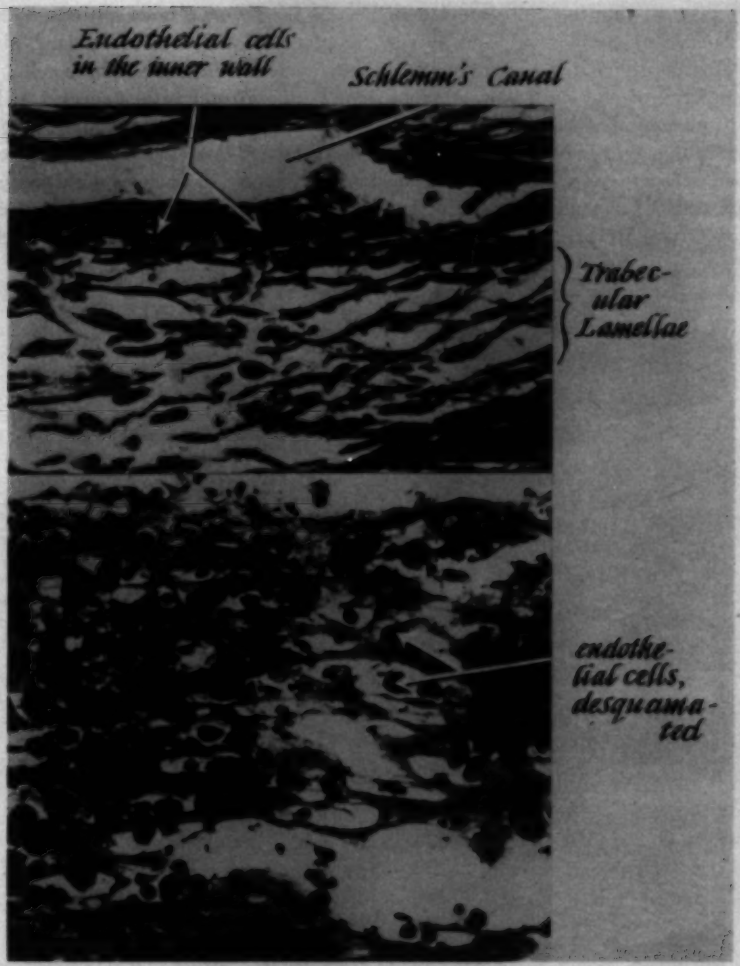

Fig. 3 (Rohen and Unger). (Above) Cross section of Schlemm's canal with adjacent meshwork in case of primary glaucoma with strong endothelial reaction. At the bottom of Schlemm's canal one may observe conglomerations of desquamated endothelial cells. (Below) Flat section of the same trabecular meshwork. Numerous isolated endothelial cells in the meshes of the trabecula may be recognized. ([Above] Azan stain, ×350; [below] chrome-hematoxylin-phloxin according to Gömöri).

(fig. 2). By these processes the endothelium proliferates considerably and is apparently able to fill the sponge-pore spaces to such an extent as to hamper the aqueous outflow and obliterate the inner wall of Schlemm's canal ("pore tissue," Flocks) (fig. 3). We also found such multiplications of cells in eyes with secondary glaucoma and in those with primary glaucoma.

We have demonstrated recently (Rohen

and Unger, 1957) that most probably these cells are capable of vital storage and, therefore, have to be designated as retothelia of the angle (RES of the eye) (fig. 2). The storage of pigment has been known for a long time. With siderosis bulbi we found the well-known selective storage of iron in the endothelial cells of the meshwork. Further studies concerning storage of vital dyes are under way.

Figure 2 shows, however, that under pathologic circumstances (secondary glaucoma, inflammatory reactions) these cells, by phagocytosis of foreign substances, may increase their load, grow considerably larger, and desquamate. We also think that those cells with a storage of bacteria and foreign bodies found in the aqueous by Amsler, Huber, and Verrey, in puncturing the anterior chamber, were the retothelia of the angle.

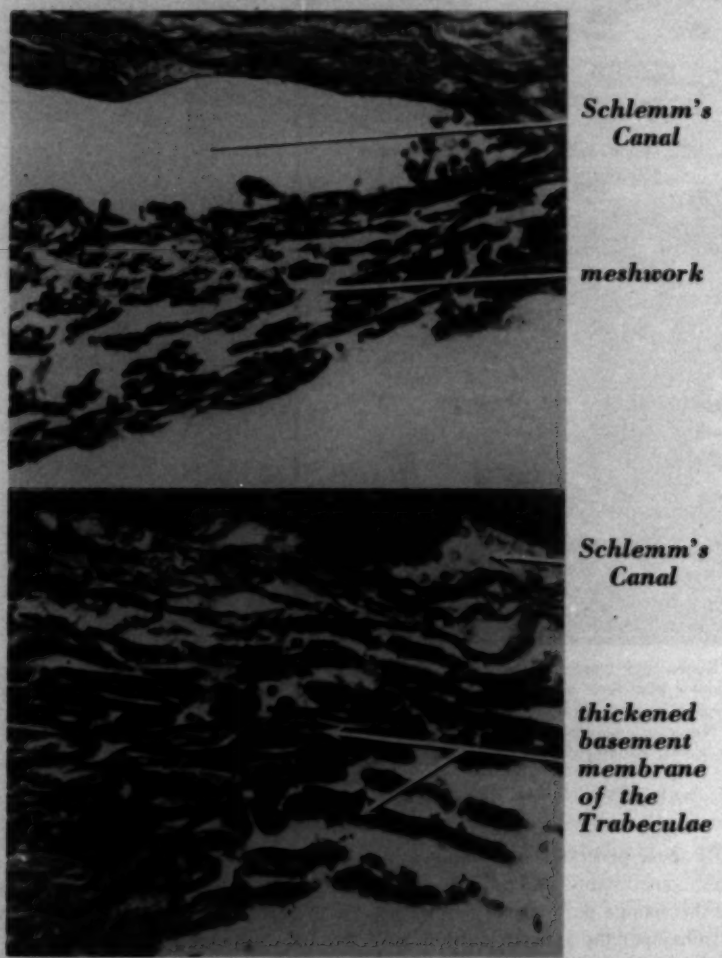

Fig. 4 (Rohen and Unger). (Above) Normal trabecula of a woman aged 49 years. (Below) Patient aged 74 years, suffering from absolute glaucoma. Note extreme thickening of trabecular lamellae.

Fig. 5 (Rohen and Unger) Flat sections of the trabecula. (a) Of a healthy, normal eye which had to be removed because of cancer of the sinus of Highmor (taken from a woman 49 years of age). (b) Of a 62-year-old patient with primary glaucoma. Strong irregular layers on the trabecular lamellae. (c) Of a patient aged 68 years. Secondary glaucoma. Almost complete conglutination of the thickened, hyalinized lamellae. Only a few oval pores are to be seen. Schlemm's canal is not visible here, yet it is still open. (All three figures, ×350.)

B. Ground substances of the trabecular meshwork

The basement membrane of the trabecula is probably in close functional dependence upon the endothelium. It is homogeneous by light-microscopy. Stained with PAS, it proves heavily reactive to periodate; however, it is not a metachromotope (Graumann and Rohen, in press). These authors found that as to its substance and structure the trabecular meshwork is characterized by

> . . . a central ground substance lamella being lined on both sides by an "endothelial basement membrane system" specialized in the metabolic process, which is well known in connection with the capillaries. The adjacent membranes in question are directly connected with the basement membrane of the corneal endothelium. Glycoproteides are part of its building substance. . . .

If these findings concerning the existence of a special basement membrane, situated between the corneal endothelium and Descemet's membrane and which communicates throughout with the trabecular meshwork, can be further corroborated (as in the case of the corneal epithelium which can be considered an established fact [Calmettes and co-workers, Graumann and Rohen]), the na-

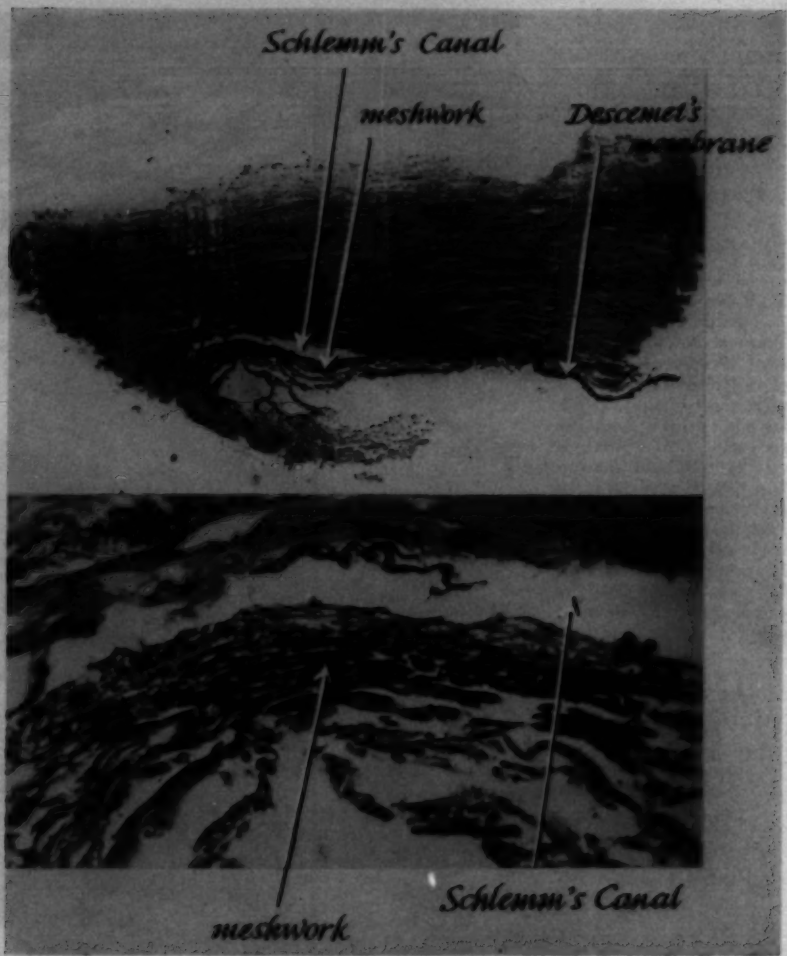

Fig. 6 (Rohen and Unger). Elliot trephination. Simple chronic glaucoma with cupping and loss of visual field. Note the thickening of the basement membrane of the trabeculae. (Orcein stain. Enlarged [above] ×80; [below] ×200.)

ture of this meshwork appears in a different light. It is, then, no longer justified to speak of a scleral meshwork (H. Virchow, 1910); rather of a modified corneal tissue or a specialized endothelial basement membrane system. Therefore, we were not surprised when, upon observing various pathologic reactions of the trabecula, especially in cases of glaucoma or perforating injuries which ended in enucleation, we could prove quantitative and qualitative alterations (Rohen and Unger, 1957), such as have been observed in other endothelial basement membrane systems of the organism, for example, due to old age, with arteriosclerosis, with follicle atresia in the ovary (Watzka, 1957), or with atrophy of the seminal tubules (H. Stieve, 1955).

2. *Pathologic alterations of the meshwork with glaucoma.* Thus we found, for example, in the eye of a woman patient, aged 74

Fig. 7 (Rohen and Unger). Flat section of the trabecular system of a child, aged four years, who suffered from perforating injury of the eye. (Azan; ×450.) Note the irregular swellings of the trabecular lamellae (arrows).

years, with simple chronic glaucoma, an enormous thickening of the basement membrane (not of the central ground substance lamella), which had caused a noticeable narrowing of the trabecular slits. By applying histochemical stains and magnifying the slides considerably under the microscope, it was discovered that layers had formed on the thickened lamellae, the former showing various histochemical reactions. In most cases the reactivity to periodate decreases as the trabecula thickens. There is no indication of metachromacy.

We rarely found this increase of ground substances in the trabecular lamellae to be as heavy as in the case pictured (figs. 4 and 5); out of the six eyes with primary glaucoma examined, all had undergone changes of the basement membrane. In 22 eyes with secondary glaucoma 14 showed no alterations, eight were altered. There were no alterations of the basement membrane in inflamed globes. Out of 14 eyes with tumors or perforating injuries, there were two cases in which the basement membrane was altered. The trabecular lamellae thicken by deposition of light-microscopically homogeneous substances, a process which may be irregular and stop at different degrees.

In Figure 5-b it can be seen that numerous lamellae have been modified while others are completely unchanged. At first the elastic fibers at the center of the lamellae are still visible, later on (fig. 5-c) they are merged in the general process of hyaline consolidation. Even histologic examination of Elliot trephinations, that is, a biopsy of glaucomatous eyes (Unger, 1956) shows an alteration of the basement membrane (fig. 6). It is interesting to note that, for example, with perforating injuries, this thickening of the trabeculae can be seen only occasionally. This causes curious shapes (fig. 7) of nodular, club-shaped swellings which are centered around a fiber lamella.

If the chamber angle is already obliterated or made impermeable by goniosynechias, one may observe especially a considerable growth and a large conglutination of the homogeneous substances. At this stage the elastic fibers are very often still well visible, whereas they disappear later on just like the endothelium itself (fig 8). We cannot decide whether it is in the endothelium or in the basement membrane that the degenerative process sets in. We have seen glaucomatous eyes in which the endothelial cells had enormously multiplied without any structural alterations of the base-

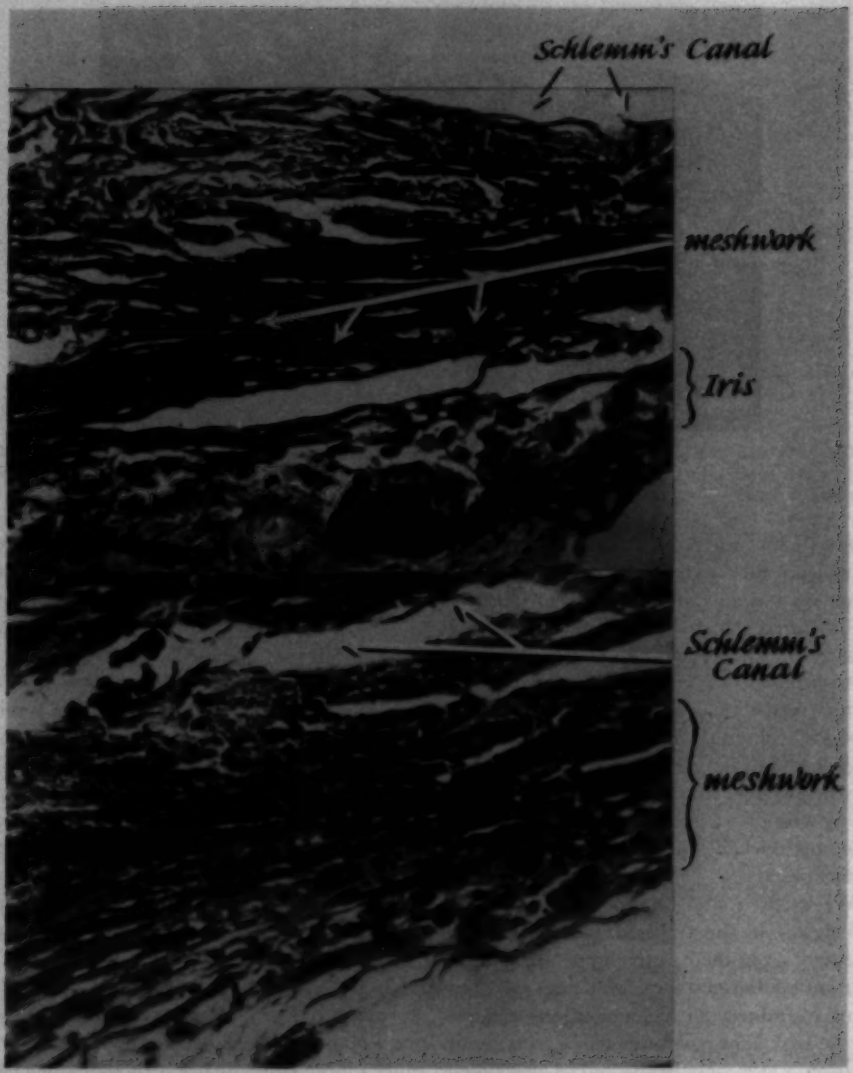

Fig. 8 (Rohen and Unger). Two cases of complete obliteration of the trabeculae by increase of the basement membrane and conglutination of lamellae in absolute glaucoma. (Above) (PAS stain according to McManus.) Note the heavily periodate-reactive coloring of the basement membrane. (Below) Cross sections of the elastic fibers still visible. (×212.)

ment membrane taking place (fig. 3), and, vice versa, a considerable thickening of the membrane without the endothelium being affected (fig. 4).

Discussion

We cannot therefore, as yet, make any definite statement as to the causal genesis of these alterations. Hence it would be pointless

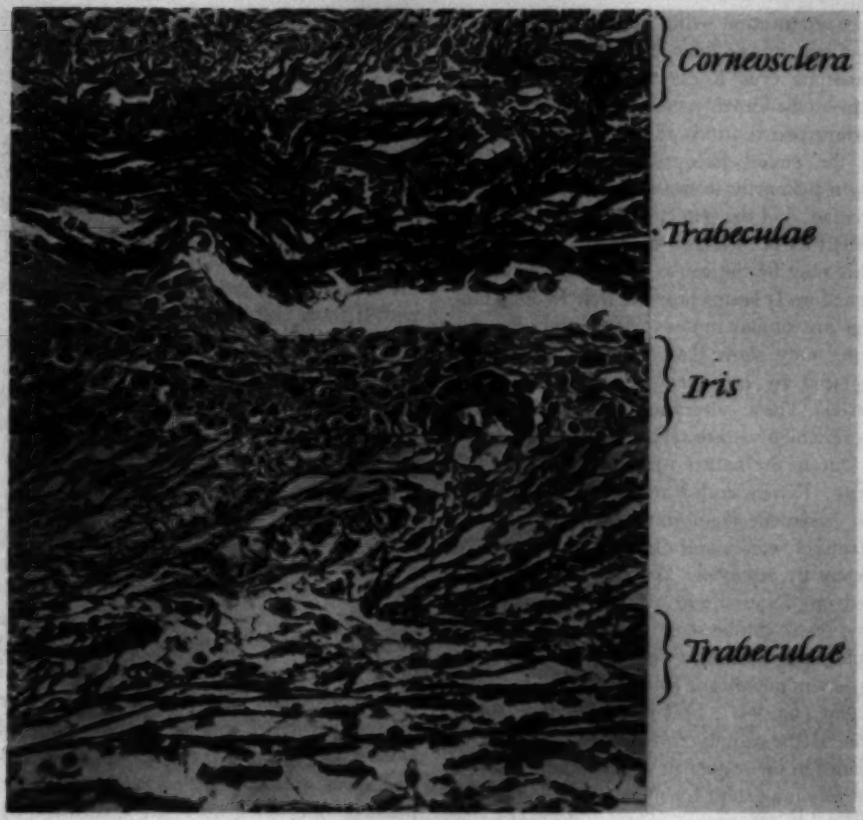

Fig. 9 (Rohen and Unger). Autolytic test. Both figures represent the same eye. (Above) Fixed immediately after enucleation. (Below) After 24 hours storage. Note complete dissolution of basement membrane by autolysis. (Both pictures ×350.)

to discuss, at this early stage of our investigation, the question whether the alterations shown are specifically glaucomatous and whether they are to be considered as primary or secondary processes.

More comprehensive investigations have been started upon. The present paper is to give some introductory information and draw the attention to a whole series of questions. Teng, Paton, and Katzin have described degenerative changes in the outermost layers of the trabecular meshwork; François, Rabaey, and Neetens, as well as Dvorak-Theobald and Kirk, recently observed a trabecular hypertrophy in eyes with glaucoma. It is, however, a well-known fact that the fibers of the meshwork become thicker and "sclerosed" with advancing years or with glaucoma.

Henderson speaks of a homogeneous substance similar to that composing Descemet's membrane, which is laid down more and more by the covering cells with each advancing decade. In some cases of glaucoma, histologically investigated, the meshwork showed a homogeneous structure with no interstices (Greeves, Polya, Sarti, Tartuferi); in other ones, the interspaces were replaced by a structureless homogeneous substance (de Vries).

In connection with his description of a chamber-lining "glass-membrane" in glaucomatous eyes Reese thought the endothelium to be capable of its production under certain provocations. This cuticular product of the endothelial cells sometimes is laid down primarily in the interstices of the trabeculae; and the tendency is for the inner lamellae to be affected more than the outer. This may be the cause of the trabecular obstruction. It seems possible that Reese's findings are similar to ours but the figures in his paper only show that the trabecular area is replaced by homogeneous tissue sparse in nuclei. These alterations are not exactly those which we are trying to describe.

Due to the nature of the material used by Teng, Paton, and Katzin (eye-bank eyes) the basement membrane disintegrates comparatively easily and disappears almost completely by autolysis. Therefore we have carried out fixation and autolysis experiments. One result can be seen in Figure 9. After the globe had been kept cool for 24 hours, the basement membrane had diminished to a large extent (fig. 9-b). This fact is another indication of the affinity to other basement membranes in the organism where solubility is extremely important for metabolism and permeability. If one considers the main part of the meshwork as an "endothelial basement membrane system," the genesis of some symptoms might possibly be interpreted as a consequence of an alteration of this system, in which process the chemically unstable basic substance of the meshwork reacts with various effects by various degrees of solubility.

Summary

We investigated the structure of the trabecular meshwork and some of its pathologic reactions. It was to be seen that the equatorial elastic reticulum which represents the mechanical basis of the trabeculae changes very little in case of disease, while the endothelium and the "glass membrane" connected with it show characteristic reactions.

The endothelium is capable of vital storage and hence ought to be designated as retothelium. The cells are able to increase, multiply, and desquamate, with structural alterations becoming evident.

The "glass membrane" is comparable to a basement membrane and can react by an increase of substance in various cases of glaucoma, as well as in globes enucleated because of tumors or perforating injuries. In eyes with absolute glaucoma this process reaches extreme degrees, so that in this way, too, the angle becomes obliterated. This must be distinguished from the reactions of a central ground substance lamella which envelops the elastic fibers of the trabeculae. The increase of the resistance to outflow might thus be explained morphologically. In agreement with Graumann and Rohen we suppose the existence of an "endothelial basement membrane system" for which laws similar to those for the wall of the capillaries may be valid.

Schumannstrasse 14.

References

Amsler, M., Huber, A., and Verrey, F.: L'humeur aqueuse et ses fonctions. Paris, Masson & Cie., 1955.

Ashton, N., Brini, A., and Smith, R.: Brit. J. Ophth., **40**:257, 1956.

Busacca, A.: Eléments de gonioscopie normale, pathologique et expérimentale. São Paulo, Rossolillo, 1945.

Calmettes, Deodati, Planel, and Bec: Étude histologique et histochemique de l'épithélium antérieur de la cornée et de ses basales. Arch. Ophthal., **16**:481, 1956.

Flocks, M.: Arch. Ophth., **56**:708, 1956.

François, J., Rabaey, M., and Neetens, A.: Arch. Ophth., **55**:488, 1956.

Graumann, W., and Rohen, J.: Chemohistol. Befunde am menschlichen Auge (Cornea, Sklera, Uvea), Ztschr. mikrosk.-anatomische Forschung, in press.

Greeves, R. A.: Proc. Roy. Soc. Med. London, Sect. Ophth., **7**:112, 1913/14.

Henderson, T.: Ophth. Rec., **17**:534, 1908.

———: Glaucoma, London, Edw. Arnold, 1910, pp. 34-38.

Kurus, E.: Vers. d. Vereinigg. Rhein-Main. Augenärzte, Frankfurt/M. 1./2. 12. 1956.

Polya: Ungar. Beiträge, **2**:319, 1899. (Quoted by de Vries.)

Reese, A. B.: Am. J. Ophth., **27**:1193, 1944.
Rohen, J.: Die funktionelle Gestalt des Auges und seiner Hilfsorgane. Verl. d. Akad. d. Wissensch. u. d. Lit., Mainz. Komm. Wiesbaden, Verlag Franz Steiner, 1953.
———: Ophthalmologica, **131**:51, 1956.
———: Ber. 60. Zusk. Deutsch. Ophth. Ges. Heidelberg. München, J. F. Bergmann, 1956, p. 50.
———: Arch. f. Ophth., **158**:310, 1957.
Rohen, J., and Unger, H. H.: Anat. Anz., **104**:287, 1957; **105**:93, 1958.
Sarti, V.: Bull. sc. med. Bologna, **4**:147, 1893. (Quoted by de Vries.)
Stieve, H.: Der Einfluss des Nervensystems auf die Geschlechtsorgane. Stuttgart, Thieme, 1955.
Tartuferi, F.: Giorn. Acad. med. Torino, **30**:624, 1882.
Teng, C. C., Paton, R. T., and Katzin, H. M.: Am. J. Ophth., **40**:619, 1955.
Theobald, G. D., and Kirk, H. Q.: Am. J. Ophth., **41**:11, 1956.
Unger, H. H.: Klin. Wschr., **34**:927, 1956.
———: Deutsch. Ophth. Ges. Heidelberg, München, J. F. Bergmann, 1956, p. 59.
———: Klin. Wschr., **35**:199, 1957.
———: Arch. Ophth., **158**:509, 1957.
———: Klin. Monatsbl. f. Augenh., **131**:385, 1957.
———: Klin. Monatsbl. f. Augenh., **131**:692, 1957.
Unger, H. H., and Rohen, J.. Ber. gi. Zusk. Deutsch. Ophth. Ges. Heidelberg, München, J. F. Bergmann, 1957, p. 256.
Virchow, H.: Graefe-Saemisch, Handbuch Augenh., Leipzig, Engelmann, 1910, v. 1, p. 281.
Vrabec, F.: Ophthalmologica, **128**:359, 1954.
———: Brit. J. Ophth., **41**:20, 1957.
de Vries, W. M.: Nederl. Tijdschr. v. Geneesk., **43**:1688, 1907.
Watzka, M.: D. Ovarium, Handb. mikrosk. Anat. d. Menschen. Bargmann, v. Moellendorf, 1957, Suppl. of VII/1.

NEWER OPTICAL AIDS FOR CHILDREN WITH LOW VISION*

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A major campaign of the National Society for the Prevention of Blindness stresses the theme that all persons, especially children, must be given every opportunity to utilize fully whatever sight they possess. Of the 33.5 million in the school popluation of the United States, 8.5 million require eye care for adequate far and near vision. Sight-saving classes for children with a correctible acuity of only 20/70 to 20/200 have an enrollment of 60,000.

These partially seeing children respond well to appropriate methods of visual education. Our recognition of their special educational status is due in great part to the late Mrs. Winifred Hathaway whose unique monograph, "The Education and Health of the Partially Seeing Child," published posthumously in 1955, is a fitting memorial of a life devoted to this cause.

Approximately five percent of the legally blind in this country, or about 15,000, are under 20 years of age. Of these 60 percent have a little sight, which ranges from vague light perception to 20/200 in the corrected better eye. Amblyopia, or uncorrectible poor vision, is more prevalent in rural than in urban areas; and in Negroes, Mexicans, and Indians than in white children. Among Negroes the ratio is at least double that in the white population.

At present about 60 percent of the amblyopia in children is of prenatal origin. Prematurity by itself, even when not resulting in retrolental fibroplasia, frequently determines amblyopic myopia. Infective diseases account for over 20 percent; accidents for about 10 percent. The danger from accidents cannot be overemphasized; more children from one to 14 years of age die from accidents than from all known childhood diseases.

Most students with normal intelligence and motivation who have sufficient vision to walk

* From the Department of Ophthalmology, Northwestern University Medical School. Read before the International Council for Exceptional Children, Kansas City, Missouri, April 9, 1958.

about unaided, that is 4/200 or better and a fair peripheral field, can be fitted an optical aid with which they can read. A young reader who holds the print near his nose is not necessarily highly myopic. Amblyopia may be the handicap. The short range produces magnification since the retinal image at a three-inch distance is five times larger than that at 15 inches. In essence this is the principle of spectacle magnifiers which simply enable one or both eyes to focus sharply at a certain short range. Magnification is obtained by merely reducing the reading distance. Children seldom have the rigid habits of elderly persons and hence have less difficulty in adjusting to a very close focus. Because of their available accommodation less spectacle magnification is required to attain the desired near acuity so that the simpler and less expensive aids are usually effective. Those already trained in braille, however, have but little inclination to struggle with visual aids.

In screening workers at The Chicago Lighthouse, who had been educated in schools for the blind, several were found to have enough vision to use optical aids effectively but nearly all preferred to continue with their familiar braille. Optical aids should not be fitted unless the ocular pathology is reasonably stationary. Progressive cataract and diabetic retinopathy, for instance, are ordinarily definite contraindications.

Improvement of distant vision

The conduction of a clear image to the retina may be impeded by errors of refraction or affections of the ocular media. The pinhole disc can usually differentiate these conditions from the perceptive type of visual impairment. The pinhole enhances the acuity in a conductive impairment that is not too diffuse or extensive, but it worsens the vision in perceptive deterioration. Should the poor sight be due to an anomaly of refraction, an adequate correction provides better vision than that through the pinhole, owing to the greater amount of light entering the eye. Every amblyope should be given a careful refraction as not infrequently the benefit thus obtained is surprising. Amblyopic vision requires that the test distance be shifted to 10 feet and extra large letters ranging from 20/800 down are often of service. Those using a mirror on a stand can obtain the 10-foot distance readily by moving the mirror. An imperfect correction lessens the efficiency of a magnifying device since the blur effects of astigmatism are likewise enlarged by magnification.

In certain corneal affections, such as conical cornea, discrete opacities, and irregular astigmatism after injury, though the pinhole improves acuity, no spectacle lens may be of value. In such cases the contact lens may often restore excellent vision. Multiple-pinhole spectacles are a less efficient alternative because of the reduced brightness and the disturbing light mosaic produced by the pinhole pattern. In unilateral aphakia, the contact glasses permit binocular vision and avert the divergent squint that may otherwise follow. Contact glasses are also of particular value in high myopia; with them a myope of −20D. gains 45-percent enlargement of the image, a fuller field, elimination of corneal astigmatism, and avoidance of the prismatic and peripheral distortions of strong spectacles.

Telescopic spectacles have serious limitations, but there is no other device that can aid distant vision regardless of the kind of ocular pathology.[1] The observer must maintain a stationary position since telescopic magnification is accompanied by a corresponding magnification of motion and reduction of field. Low-power telescopes, such as the comparatively inexpensive Lamont or Wollensack sport-glasses, are helpful at school for seeing the blackboard and watching demonstrations; and at the theater or cinema. They are unnecessary for viewing television as adequate magnification is obtained by getting close enough to the screen. Though a person with 8/200 vision and an

intact field can travel unaided, he cannot read street signs or identify buses. For this purpose a pocket telescope is useful, such as the six-power penscope. The field is only eight degrees but it is large enough to following moving objects if held in a steady hand.

Aids for near vision

For aiding near vision five general types of magnifying devices are available; hand magnifiers or loupes, high plus additions, telescopic units, aplanatic magnifiers wedged in a spectacle lens, and apparatus for projection enlargement. The amplification should be no greater than is required for the person's predominant need, as increasing magnification reduces inevitably both reading distance and field.

Loupes

A convex lens is the simplest form of magnifier and is widely used as a hand reading glass.[2] The best design is plano-convex with the flat face toward the eye. The useful field seldom exceeds 10 to 12 degrees. A reading lens with a suitable hyperbolic surface is now available, molded in clear plastic, which covers sharply a much larger field than its spherical counterpart. These aspheric plastic magnifiers, obtainable in various strengths from 4.5 to 20D., have the incidental advantages of light weight and high resistance to breakage. Magnifiers stronger than 10D. should have a supporting device to maintain a fixed distance. Children secure a greater magnification when the adjustment requires use of the accommodation as in the four-times Plasta cataract reader for which the elderly must use their presbyopic correction. A loupe with a thread screw can compensate simple refractive errors by screwing the lens down for myopia or up for hyperopia. Magnifiers with built-in illumination, supplied by a battery, are especially helpful in perceptive depression.

In computing magnification the amplified image is compared to that at the nearest point of distinct vision of the normal eye, which has been arbitrarily set by optical physicists at 10 inches or 4.0D. vergence. According to this convention the enlargement produced by a magnifier is found by dividing its dioptric power by four, or dividing 10 by its focal length in inches. Conversely the focal length in inches is determined by dividing 10 by the magnification. Thus a five-times Hastings lens has a focal distance of two inches. The diameter of the field of view is always somewhat less than the focal length. Depth of focus refers to the range that a magnifier can be moved toward or away from an object and still maintain a sharp image. Depth of focus, reading distance and field of view shrink simultaneously as the power increases.

Spectacle magnification

Any lens or lens combination employed as a loupe can be mounted in a frame and used for spectacle magnification. As long as the object is precisely at the anterior focal point, increasing the distance of the lens from the eye reduces somewhat the field but does not compromise the image. Hence the additive power can be attached to a headband, as in the Magnifocuser, or secured by a swivel clip to the distance lens of the better eye, as in Behr's spectacle loupe, an inexpensive device commonly used by watchmakers. Behr's spectacle loupes can be rotated readily in or out of position and are available in focal lengths of six to 1.5 inches.

In bilateral amblyopia of moderate degree both eyes may be fitted with plus additions, up to 5.0D., as better vision, a larger field, and greater depth of focus result from binocular vision.[3] The increased convergence can be relieved by setting the lenses or bifocal segments at less than the true interpupillary distance.

For monocular vision high plus additions of eight, 16, 24, and 32 diopters are now available. High plus additions have the advantages of simplicity, economy, and conventional appearance and are to be preferred when applicable. Aspheric glass and plastic lenses are now marketed that give a larger

effective field at high magnifications than spherical lenses.* The difficulty of illumination at a close reading distance—when the print is in the shadow of the face—can be overcome by a light attached to the frame and supplied by a battery held in the pocket. An attachment to the frame can also prefix the lens-to-object distance so that a sharp focus is steadily maintained.

Telescopic aids

Several types of telescopic aids for near vision are available.[4] The cheapest is a monocular appliance of twofold magnification that can be clipped over the spectacle lens of the better eye. In the Kollmorgen instrument the distance correction and the reading addition snap on to the rear and front of the telescopic unit respectively, thus adding flexibility but also weight to the apparatus. Telescopic spectacles for near use of ×3.5 magnification have a field of two inches, the width of the narrow newspaper column found in the tabloids. With higher magnification the field is proportionately less.

In the Univis telescopic spectacle lens, a Steinheil cone is inserted through the distant correction at the position that a bifocal segment occupies. This gives the spectacles a bizarre appearance but also several advantages—lightness in weight, adaptability to any frame, and a portal for distant vision above the telescopic cone. The Bechthold and Feinbloom telescopic spectacles are air-spaced compound lens systems in which lightness is achieved by using plastic lenses and incorporating the refractive correction into the near lens.

Triple aplanats

The triple aplanat, popularly known as the Hastings lens, is a cemented triplet consisting of a biconvex lens of crown glass between two negative lenses of flint. It gives a sharp field that is free from chromatism and distortion. A seven-power Hastings lens has the same magnification as a simple 28D. lens but without the distortion that limits the resultant field. Compared to a telescopic combination of similar power, the Hastings lens gives a larger field but at half the reading distance. Like the Univis telescopic unit, the triple aplanat is inserted completely through the lens at the patient's usual reading level. Triple aplanats are available in three to 20 power and are cheaper than a comparable telescopic device. For the sake of lightness a plastic carrier lens is used.

The triple aplanat is the most effective device for aiding severely amblyopic vision. A young woman with bilateral central scotomas and a visual acuity of 10/200 in each eye reads five-point at three inches with a ×5 triple aplanat, which she prefers to the Feinbloom telescopic spectacles previously worn. She is working as a full-time typist at The Chicago Lighthouse. A man with Leber's optic atrophy and 6/200 acuity in his better eye reads five-point at three inches with a seven-power triplet and is thus able to follow specifications in the workshop. Usually after a month's practice a book page can be read in half the time than when the aid was first used.

Projection enlargement

Magnification of opaque material by projection from a Balopticon is familiar in the classroom. In an adaptation of this principle reading material is projected on a screen similar to that in the familiar television set.

* Ronchi, in *Optics: The Science of Vision*, translated from the Italian into English (1957), questions whether amblyopes can appreciate the improved image that results from a more perfect lens. My clinical tests, designed to test this point show that they do. A typical example concerns a veteran of the Korean conflict, who suffered from a mine explosion, bilateral corneal opacities, and cataracts. After a corneal transplant in one eye, followed later by removal of the cataract, he was fitted with a +32D. add with which he could read six-point type except when the contrast was poor as in a folder with black type on brown paper. With a +30D. Volk conoid lens, he could read this folder and even four-point type in the newspaper. The Volk conoid lens, however, cannot be prepared as a bifocal. Recently the American Optical Company has developed "Aolite" aspheric lenses in scratch-resistant plastic with ×8, ×10, and ×12 magnification that are of lighter weight and lower price.

The model evolved by the Franklin Institute enlarges ×3 and ×5 and is useful for those with vision above 15/200. The Megascope, sponsored by the American Foundation for the Blind, magnifies ×12 and ×25 and has helped some cases with vision as low as 4/200.[5] The magnification figures are for viewing at 16 inches; at half that distance the magnification is doubled. Projection magnification is unsuccessful when the light sense is much diminished, as in optic atrophy.

Clinical survey

Optical aids find their greatest usefulness in helping near vision.[6] Generally, the most serviceable are those prepared as bifocals. They are apparently valueless if the corrected acuity is below the counting of fingers at four feet. Above this minimum 50 to 75 percent of the severely amblyopic can be assisted. When more than twofold magnification is necessary the desired goal is often best achieved gradually. More than one aid may be required. A student was prescribed originally telescopic spectacles with which he saw the blackboard better and with the near addition read ordinary textbooks. He was later fitted with a ×10 triplet so that he could read when necessary the finer print of footnotes and references.

Though an adequate optical correction of refractive errors renders the measurements of far and near visual acuity parallel, the two measurements are not identical—near vision being slightly less acute—as accommodation and fixation are usually adjusted more precisely for distance than for near.[7] However, near vision may be better than that for distance in cases of peripheral opacities of cornea or lens, irregular astigmatism, or pendular nystagmus.[8] Following a battery fluid explosion, a youth required enucleation of one eye and the other eye after healing had scattered corneal opacities, irregular astigmatism, and an unimprovable 20/200 vision. Nevertheless, a ×1.7 telescopic unit with a reading addition of 4.0D. allowed him to read four-point type, the average normal near acuity. The vision for near is markedly poorer than that for far in the presence of a central opacity of the cornea or lens or of an incomplete central scotoma.

A minimal requirement for near vision is ability to read newspaper or six-point type. The 1955 official formulation of visual efficiency revalues radically the disability incurred by poor near vision. While the visual efficiency assigned to six-point type is 90 percent, that allotted to eight-point is now only 50 percent. Testing of low vision is facilitated by the new transilluminated near vision kit in which any variety of small slides can be inserted. By this means the brightness of the print remains constant regardless of the distance from the eyes. The apparatus has the interest of novelty while the small slides stimulate attention. As the semisighted need all the clues they can get, running text is to be preferred,[9] though tests that demand no literacy are also included.

In small macular lesions the spreading effect of magnification often gives a better acuity than would be anticipated. As the field of view is reduced by every magnifying device, these visual aids are not too helpful in advanced cases of glaucoma, optic atrophy, or retinitis pigmentosa where the visual field is already very contracted. Since a marked diminution of contrast occurs with partial cataract, much magnification is not then acceptable as the contrast is further reduced thereby.[10] In these cases a reading slit in black cardboard is a useful accessory; it helps to keep the place, concentrates the attention, and masks off the light from nearby brighter areas. In partial cataract, the reading slit by reducing the veiling glare often effects a remarkable improvement in reading vision.

The past few years have witnessed a revival of interest in optical aids, significant new developments in this field, and the establishment of many visual aid centers in this country and abroad. Such projects are now active in New York, Brooklyn, Philadelphia, Baltimore, Pittsburgh, Chicago, St.

Louis, Los Angeles, Oklahoma City, and Charlotte, North Carolina. To determine what aid is best for a particular patient requires experience and judgment. Success demands patience and confidence from both the subject and the examiner.

Of the last 200 cases examined at The Chicago Lighthouse, having uncorrectible acuity of 20/80 to 5/200, about 75 percent were enabled to read six-point type or better. One student with congenital amblyopia and pendular nystagmus had previously transcribed in braille and followed his assignments through the service of a reader. Since using the optical aid, a ×10 triple aplanat, he now does his own reading. Another student with bilateral familial macular degeneration was fitted with a Wollensack three-power binocular telescope and a ×10 triplet. His mother sent the following acknowledgment:

"Stuart writes that the binoculars help him to see the drawings and writing on the blackboard. The reading glasses enable him to look up words in the dictionary for himself and read his own mail as well as his textbook assignments. Thank you for all that you have done."

Another case of bilateral familial macular degeneration responded well to bilateral adds of 4.5D., and his academic average rose from C- to B. A visual aid enabled a youth with retinitis pigmentosa to study music and realize his desire of performing in the church choir.

One more case should be cited:

A six-year-old child with retrolental fibroplasia had no vision in one eye and in the other uncorrectible 20/100 acuity. With a +8.0D. bifocal add he read four-point in the illiterate chart. He entered school with normal-sighted children and was given, as advised, front seat placement. His parents report that his progress has been entirely satisfactory.

Amblyopic myopes of high degree, though seeing poorly with their distance correction, often read fairly well without glasses. Such patients can be fitted advantageously with what one may call reverse clerical glasses—rimless spectacles with the lower part of each lens cut off three mm. below the center. The following case illustrates a typical indication for this special form:

A nine-year-old child with amblyopic myopia and pendular nystagmus, caused by prematurity and over-treatment with oxygen, required −10.0D. sph. bilaterally with which her acuity was barely 10/200. Without glasses, she read five-point type at four-inch distance and achieved excellent grades in the regular classes of the public school.

Forty years ago our educational systems had no alternative for partially seeing children but the braille schools where instruction proceeded by necessity at one-fourth the rate of normal-sighted children. The advent of the sight-saving class changed the picture and enabled such children to approximate normal progress. As the cases cited illustrate, the education of the semisighted may be aided greatly by optical devices, and their use can expand considerably the number of children adaptable to visual education. In any individual case a prognosis of the long-range value of an optical aid cannot be made entirely on the basis of visual acuity and ocular pathology since intelligence and background, perseverance, and motivation are nearly of equal importance.

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References

1. Tait, E. F.: Optical aids in severe visual disability. Survey Ophth., **1**:205-220 (June) 1956.
2. Linksz, A.: Visual aids for the partially sighted. Am. J. Ophth., **41**:30-41 (Jan.) 1956.
3. Fonda, G.: Binocular corrections for low vision. Am. J. Ophth., **45**:23-27 (Jan.) 1958.
4. Lebensohn, J. E.: Telescopic spectacles. Sight-Saving Rev., **21**:204-21, 1951.
5. Gordon, D. M., and Ritter, C.: Magnification. Arch. Ophth., **54**:704-716 (Nov.) 1955.
6. Lebensohn, J. E.: Optical aids for subnormal vision. Sight-Saving Rev., **28**:201-209, 1956.

7. ———: Visual acuity tests for near. Am. J. Ophth., **45**:127-133 (May, Pt. II) 1958.
8. Kestenbaum, A., and Sturman, R. M.: Reading glasses for patients with poor vision. Arch. Ophth., **56**:451-470 (Sept.) 1956.
9. Sloan, L. L., and Habel, A.: Reading aids for the partially blind. Am. J. Ophth., **42**:863-872 (Dec.) 1956.
10. Stimson, R. L.: Optical Aids for Low Acuity. Los Angeles, Braille Inst. of Am., 1957.

EVALUATION OF A MODIFIED BLASKOVICS OPERATION (ILIFF TECHNIQUE) FOR BLEPHAROPTOSIS*

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From 1954 through early 1957, 115 modified Blaskovics operations based on the Iliff technique for ptosis had been performed at various Detroit hospitals. Their evaluation is of interest, since there has been no previously published study of this size on results with the Iliff modification. Since Dr. Iliff described his technique in 1954,[1] it has become a favorite choice of many surgeons for ptosis with levator action. The usual reviews of ptosis classifications and operations have been omitted from this paper, since they are adequately covered in literature.[2-8]

It should be emphasized that Dr. Verhoeff originated this type of modified Blaskovics operation before 1930.[9] The fact that he did not report it in a formal publication does not detract from the credit due him. There are others, particularly Dr. Guyton,[10] who have been using this operation for many years before Dr. Iliff's report. Despite these priorities, we concur with the title "Iliff modification," since it is already in common use, and Dr. Iliff developed this modification independently, published it first, and has popularized it.

* Presented at the 16th annual meeting of the Wilmer Residents' Association, Baltimore, April 5, 1957. The cases reported herein were collected from the Department of Ophthalmology, Henry Ford Hospital, and from Children's Hospital, Providence Hospital, and Receiving Hospital of Detroit.

OPERATIVE TECHNIQUE

All of the 115 modified Blaskovics operations included in this series had a block resection of the levator and tarsus with conjunctiva as in the Iliff modification. The general technique of the operation and its variations as employed by us is reviewed herewith:

The conjunctival route is always used. The upper lid is everted with a lid retractor (fig. 1A). With sharp-pointed scissors, buttonhole stab incisions are made just medial and lateral to the superior edge of the tarsus to enter the potential space between the upper segment of the tarsus and the septum. One jaw of a ptosis clamp (either a Berke or Iliff type clamp, or a small, curved hemostat) is passed into the temporal buttonhole, then passed across the lid beneath the upper border of the tarsus, and out through the nasal buttonhole (fig. 1B). The jaws are clamped together. Usually, there is no marked resistance to the passage of the clamp between the septum and the upper segment of the tarsus. Marked resistance to passage of the clamp can be relieved by inserting blunt-tipped scissors in each buttonhole and bluntly dissecting toward the center of the lid between skin and tarsus. An incision is made distal to the clamp, through the tarsus but relatively close to its upper border. Hemostasis will have been achieved largely by virtue of having clamped the superior portion of the tarsus.

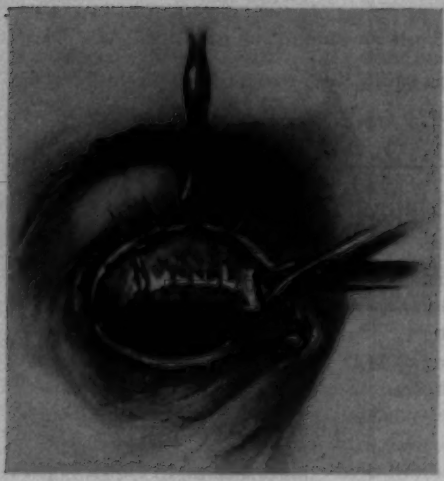

Fig. 1A (Schimek and Cusick). The upper lid has been everted with a lid holder. With sharp-pointed scissors, buttonhole incisions are made medial and lateral to the superior edge of the tarsus to enter the potential space between the upper segment of the tarsus and the skin.

Fig. 1B (Schimek and Cusick). One jaw of a Berke ptosis clamp has been passed into the temporal buttonhole, passed across the lid between tarsus and skin, and out through the nasal buttonhole. The jaws are clamped together. An incision is made distal to the clamp through the tarsus close to its upper border.

Fig. 1C (Schimek and Cusick). By sharp and blunt dissection the levator, Mueller's muscle, and the conjunctiva are separated as one layer from the lid and septum orbitale toward the level of the upper fornix.

In lids which have had previous levator and tarsal resection, it usually is desirable to preserve the remaining tarsus, limiting the resection to the levator. In these, buttonhole stab incisions are placed so the clamp is passed beneath the junction of levator with tarsus. Passage of the clamp generally is easy and hemostasis good, even in these instances. If desired, a partial tarsectomy can be performed later in the operation as a separate step (as in the original Blaskovics operation) before the placement of sutures.

By sharp and blunt dissection, the levator, Mueller's muscle, and conjunctiva are separated as one layer from the lid and orbital septum (fig. 1C). At this stage of the operation, technique may vary. In the majority of cases in this series, a minimum of dissection was performed with no cutting of the levator horns (as in the original Iliff technique). In this majority, a levator resection of 10 mm. was the average for cases of congenital ptosis. A minority of cases had a more extensive dissection of the levator with cutting

of the medial and lateral horns. In this minority, the average amount of levator resection varied from 12 to 18 mm.

In all cases, double-armed sutures are passed through conjunctiva and levator at a measured distance from the cut border of the tarsus (fig. 1D). Four or five double-armed sutures are routinely used. The levator is resected two mm. in front of the suture line. The sutures are passed through the lid anterior to the tarsus to emerge about four mm. above the lashline (fig. 1E) and are tied. In this series, either catgut (P. L. C.) or silk (R. A. S.) was used. In those cases which had minimal dissection around the levator (the majority), if the ptosis was moderate and levator action good, the sutures were not passed through the orbital septum; but in those with marked ptosis and poor levator action, the sutures included a bite through the orbital septum about four mm. back of its edge (P. L. C.). The latter technique utilizes the elastic tension of the septum to help elevate the lid. In the minority of cases with extensive dissection around the levator (and greater amounts of resection), the orbital septum was not shortened by the sutures regardless of the degree of ptosis.

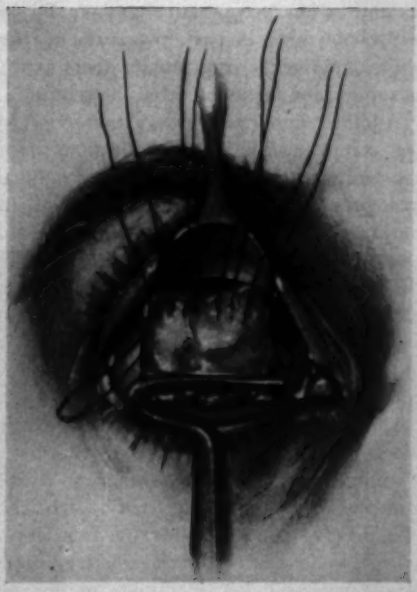

Fig. 1D (Schimek and Cusick). Four double-armed sutures are then passed through the layer of conjunctiva, Mueller's muscle, and levator about 12 to 16 mm. from the free border. Excision is performed two mm. in front of the suture line.

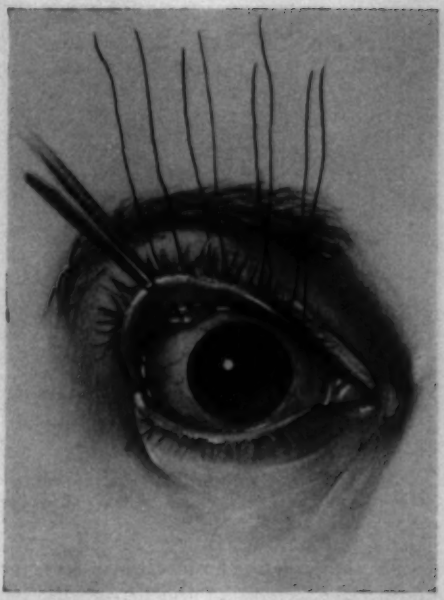

Fig. 1E (Schimek and Cusick). The sutures are passed through the lid anterior to the tarsus to emerge about three to four mm. above the lash line and are tied.

The routine use of Frost sutures to hold up the lower lid and protect the cornea is not believed to be necessary. Separate lid fold sutures also are not necessary in the usual case. The eye is either dressed with some pressure, or more frequently simply protected (without dressing) by means of a Fox shield. The patient is usually discharged from the hospital on the following day, the lid sutures being removed in five to seven days.

The sutures through the levator and lid can be tied over individual glass beads, rubber strips, or cotton pegs, or over a single, long, rubber strip. One of us (R. A. S.) prefers to loop the sutures through and tie them over cut sections of rubber catheter, as shown in Figure 1F. In case an overcorrection or asymmetrical correction is apparent

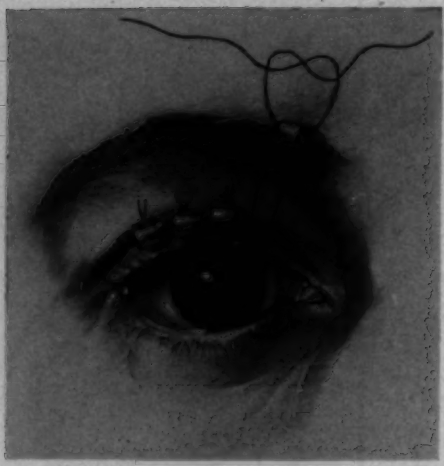

Fig. 1F (Schimek and Cusick). The sutures may be looped through and over cut sections of rubber catheter and tied.

during the first few postoperative days, the appropriate sections of rubber catheter can be cut and removed, and the sutures (still intact) allowed to retract about four to six mm. into the lid. Downward massage of the lid will loosen the levator attachment somewhat (after the sutures are loosened or removed) during the first three or four days after operation. To loosen the levator attachment after more firm healing, the lower tarsus must be pulled forcibly downward. (Under local anesthesia, a good grasp along the lid border can be obtained with a small, chalazion clamp or a ptosis clamp.)

General observations

Some broad generalizations can be made on this series without a detailed statistical analysis. The average postoperative Iliff type operation requires a certain amount of time —usually two to three weeks—to approach the final result. During the first week, the lid should still be somewhat ptotic. As edema and swelling are disappearing, the lid will retract upward during the first one to three weeks after operation. As with other levator resection methods, a postoperative lid lag will usually be present in congenital ptosis with poor levator action. As the postoperative period lengthens, the lid lag will improve to some extent. In regard to this latter observation, Dr. Iliff stated in his article, "Since the septum has not been shortened, no checking action to downward gaze is encountered." In our series, however, some lid lag was apparent on downward gaze whether or not sutures passed through the orbital septum, particularly in the congenital ptosis cases with poor levator action preoperatively. Berke and Wadsworth[11] have shown that congenital ptosis often has levator muscle partly replaced with connective tissue. Some lid lag will necessarily result when a partially fibrotic levator is shortened sufficiently to obtain a good result. It should be remembered that cases of congenital ptosis often show some lid lag on extreme downward gaze even before any surgery.

Those cases which had the septum included in the sutures usually show a marked lid lag in downward gaze and a mild ptosis in upward gaze. Since these cases all had poor levator action preoperatively, their lid lag is probably due in part to a more marked fibrosis of their levator muscle fibers as well as some splinting action from the septum.

Another important observation concerns the complete lack of complications from shortening of the conjunctiva. Many ophthalmologists protest against resection of the conjunctiva in block fashion with the levator muscle. Their fear of some future need for the resected conjunctiva would seem reasonable. However, after the overlying levator has been shortened by the classic Blaskovics operation, the preserved conjunctiva is in excess and probably shrinks to conform with the underlying scarring. Furthermore, with the Iliff operation, subconjunctival scarring is minimal, and the conjunctiva readily stretches to such an extent that no deficiency is clinically apparent. Thus, the final difference in the extent of the conjunctiva, after a classical Blaskovics operation on the one hand and an Iliff on the other, probably is not as great as is usually alleged. There has

been no apparent adverse effect from resection of conjunctiva in this series of 115 cases.

There has been no difficulty or complication from "dry eyes" or corneal ulcers in this series. Also, no effect on lacrimal secretion is evident, since there is minimal trauma to the lacrimal ducts.

Dr. Iliff has stated that "The secondary elevating actions of the superior rectus and the levator are enhanced by the resection of the palpebral conjunctiva, which puts stretch on the fibrous attachments from the sheaths of these muscles to the upper cul-de-sac." Whatever the correct theoretical explanation, it does seem that more elevation is obtained with the Iliff technique per given amount of resection than with a classical Blaskovics procedure, and still more than is the case when so extensive a dissection as advocated by Berke is performed. The following case seems to illustrate our experience in a reasonably fair manner: A patient with a bilateral congenital ptosis, which was less marked on the right side, had 16 mm. of levator resected by the classical Blaskovics technique on the right. The left lid with the greatest amount of ptosis had 16 mm. of levator and conjunctiva removed by the Iliff technique. Both operations were done by the same surgeon at the same sitting. The lid was raised somewhat higher by the Iliff type technique, even though this was the side with the greater ptosis originally (fig. 2).

Fig. 2A (Schimek and Cusick). Preoperative appearance of bilateral ptosis, more marked left eye, with fair levator function, OU.

Fig. 2B (Schimek and Cusick). Postoperative appearance with Blaskovics operation performed on the right and the Iliff technique on the more severely drooping left upper lid. The same amount of levator was resected in each eye but the Iliff technique was more effective.

Our experience certainly confirms Dr. Iliff's observations on the relative ease of this operative procedure. With the clamp on the upper edge of the tarsus, there is much less bleeding, and one is spared the time-consuming and troublesome task of dissecting conjunctiva from overlying levator. What dissection is done lies between natural tissue planes, as between levator and orbital septum.

This technique is particularly valuable and easy to perform when repeating op-

Fig. 3 (Schimek and Cusick). Left unilateral congenital ptosis of moderate degree operated by the Iliff modification. The left column of pictures shows the preoperative appearance on looking up, straight ahead, and down. Note that the ptotic left upper lid lags somewhat in downward gaze. Center pictures show the result two days after an Iliff resection of the levator and conjunctiva. It is typical of a good result that there is still some ptosis in the primary position due to swelling and edema of the lid during the first postoperative week. Also, there is lid lag on looking down with the operated lid not following down as well as the unoperated lid. The right column of pictures show the appearance two weeks after operation. The operated left upper lid still has increased lid lag. This lid lag tends to diminish during the subsequent postoperative period. This was classified as an excellent result.

erations on previous unsuccessful levator resections. Another general observation was that results with the Iliff modification seemed better to us than results in cases operated either with the classical Blaskovics technique or with Berke's modifications. (The principle of extensive dissection of the levator in Berke's modification is largely just the opposite of the principle of the Iliff modification.)

Fig. 4 (Schimek and Cusick). Old traumatic ptosis of left upper lid due to perforating injury which cut the levator insertion. The left column of pictures shows the preoperative appearance on looking up and straight ahead with levator action present but reduced. Center pictures show the appearance two days after a resection of the levator by the Iliff technique with reattachment of the levator to the tarsus. The usual residual ptosis during the first postoperative week from edema and swelling is seen. The right column of pictures shows the postoperative result two weeks later with the patient looking up, straight ahead, and down. Some edema of the upper lid still was present at this time, but subsequently cleared. The final result was excellent. The lid lag on downward gaze gradually diminished.

Statistical analysis of cases

Excluded from this study are three cases with an inadequate follow-up of less than three weeks. Also excluded are one case of progressive nuclear ophthalmoplegia and one case of postencephalitic ophthalmoplegia, both of which were deliberately undercorrected to avoid corneal complications.

The remaining 115 modified Blaskovics operations based on the Iliff technique were consecutive cases performed at Henry Ford Hospital, Childrens Hospital, Receiving Hospital, and a large number of private cases (P. L. C.) performed for the most part at Providence Hospital. About one half of the cases in this series were private patients (P. L. C.) done mostly at Providence Hospital, about one-fourth of the cases were from Henry Ford Hospital, and the remaining one-fourth were distributed between Childrens Hospital and Receiving Hospital.

As shown in Table 1, 84 percent of cases were performed for congenital ptosis. Eight cases or seven percent were porformed for acquired traumatic ptosis. Ten cases or nine percent were performed for acquired nontraumatic ptosis without any association with any neurologic defect. One of the acquired nontraumatic ptosis cases may have been caused by syphilis since the patient had a positive Wassermann and an atypical unilateral keratitis, and another patient had a bilateral ptosis occurring after the age of 50 years with atrophic changes of the lids compatible with "ptosis myopathica." The remaining acquired nontraumatic ptosis cases had no apparent associated etiology and are herein designated by the term "acquired hereditary ptosis" (idiopathic).

In Tables 1 to 7, symmetric results within approximately one mm. of the desired lid level with a satisfactory lid fold were classified as excellent. Results which were 2.0 to 2.5 mm. under or over the desired lid level were classified as good and separated according to whether they were slight undercorrections or slight overcorrections. Results which were three or more mm. under or over the desired lid level were listed as poor results and classified as considerable undercorrections or overcorrections. Many of these "poor" results actually showed a marked improvement: for example, one bilateral ptosis had a marked undercorrection of both lids, but the cosmetic appearance was greatly improved, with the parents satisfied.

Fig. 5 (Schimek and Cusick). Old "traumatic" ptosis which occurred after removal of dermolipoma from the left upper lid with the insertion of the levator cut. The left column of pictures shows the preoperative ptosis with the child looking up and straight ahead. Pictures in the right column show the result two weeks after operation, with some slight amount of residual ptosis. This slight amount of residual ptosis persisted so that the final result was classified as slightly undercorrected.

In the congenital group, there were 66 percent with excellent results and nine percent with slight undercorrections, and two percent with slight overcorrections. Twenty-three percent were considerably undercorrected. There were no considerable overcorrections in the congenital group. From Table 1, it can be seen that there is little risk of overcorrection in congenital cases, and the main concern should be that a large enough resection is performed.

By contrast, Table 1 shows that the acquired cases of ptosis had a high incidence of overcorrection. Of the 10 acquired nontraumatic cases, two results were excellent; five results were good, although four of these were slightly overcorrected; and three results were considerably overcorrected and

TABLE 1*

RESULTS OF MODIFIED BLASKOVICS OPERATION BASED ON ILIFF TECHNIQUE
(115 Cases)

Type of Ptosis	No. Cases	Excellent	Good		Poor	
			Slight Under-correction	Slight Over-correction	Considerable Under-correction	Considerable Over-correction
Congenital	97	64 (66%)	9 (9%)	2 (2%)	22 (23%)	0 (0%)
Acquired hereditary (nontraumatic)	10	2 (20%)	1 (10%)	4 (40%)	0 (0%)	3 (30%)
Acquired traumatic	8	4 (50%)	1 (12%)	0 (0%)	1 (12%)	2 (26%)

* A postoperative result within 1.0 mm. of the desired lid level (approximate) was classified as excellent. If the result was 2.0 to 2.5 mm. under or over the desired lid level, the result was classified as good (slightly undercorrected or slightly overcorrected). If the result was 3.0 or more mm. under or over the desired lid level, the result was classified as poor (considerable undercorrection or overcorrection). Some of the "poor" results that are listed as considerable undercorrections had a marked improvement. The cases shown in the second row are acquired nontraumatic ptosis cases without any associated neurological defect and with good levator action. Most of this group were classified as "acquired hereditary ptosis," but one patient with ptosis myopathica and another patient with a possible luetic etiology were included.

required reoperation. Since the amount of resection averaged less in the acquired groups, the incidence of overcorrection in these groups is all the more striking. This warns that only small amounts should be resected in acquired cases.

Table 2 shows the results with acquired nontraumatic ptosis in greater detail. The two cases with a resection of less than five mm. yielded one excellent result and one slight undercorrection. Among the five cases with an eight to 10 mm. resection, there was one excellent result, two slight overcorrections, and two considerable overcorrections. Both of the considerable overcorrections required subsequent levator recessions (fig. 6). Among the three cases that had a 12 to 13 mm. resection but with cutting of the levator horns, two were slight overcorrections and one was a considerable overcorrection. In acquired nontraumatic ptosis with good levator action without any associated neurologic defect (usually classified as acquired hereditary ptosis), a resection of about five to six mm.

TABLE 2*

ACQUIRED "HEREDITARY" PTOSIS (NONTRAUMATIC) OF MODERATE TO MARKED DEGREE
(10 Cases)

Amount of Resection	No. Cases	Excellent	Good		Poor	
			Slight Under-correction	Slight Over-correction	Considerable Under-correction	Considerable Over-correction
3–5 mm.	2	1	1	0	0	0
8–10 mm.	5	1	0	2	0	2
12–13 mm. c horns cut	3	0	0	2	0	1

* Results in acquired nontraumatic ptosis cases with good levator action and without any associated neurologic defect: In this group, one patient may have had a luetic ptosis (positive Wassermann with an atypical keratitis), and another patient had atrophy of the lids compatible with ptosis myopathica. The remaining cases were classified as "acquired hereditary ptosis." As in Table 1, the excellent results were within 1.0 mm. of the desired lid level with a symmetric result and a good lid fold. The good results were 2.0 to 2.5 mm. under or over the desired lid level (slight under or slight overcorrection). The poor results were 3.0 mm. or more off from the desired lid level. There is a marked tendency to overcorrection in these cases of acquired ptosis.

Fig. 6 (Schimek and Cusick). Overcorrection following the Iliff modification for an acquired ptosis of the right upper lid of moderate degree with good levator action. The pictures in the left column show the marked overcorrection which was present two weeks after an Iliff modification was performed on an acquired ptosis of undetermined etiology (classified as "acquired hereditary ptosis"). The middle pictures show the result at one week after a recession of the levator was performed. The right column of pictures shows the final result two months after the levator recession. This illustrates the danger of overcorrection in acquired ptosis, and that correction of such marked elevation after firm healing has occurred must depend on a large recession of the levator. After such a levator recession, the lid should appear somewhat ptotic for several weeks, since the lid will usually retract upward for four to six weeks after the recession of the levator.

is a reasonable amount. This amount should be varied according to the degree of ptosis.

In Table 3 are shown the results in eight cases of acquired traumatic ptosis. Of the two cases due to blunt injury, both were considerably overcorrected with levator resections of 12 to 15 mm. respectively. Of the six ptosis cases due to section of the levator, four were excellent, one was slightly undercorrected, and one was considerably undercorrected. This suggests that there may be less danger of overcorrection if the injury causing the ptosis is perforating rather than blunt. A pertinent factor in old traumatic cases, in which the levator insertion has been cut, is that much of what one thinks is levator at the time of resection may not be actual levator muscle, so that the true resection may be much smaller than believed at the time of operation.

Table 4 analyzes the results of 77 cases of congenital ptosis with respect to the preoperative degree of ptosis. Mild ptosis includes drooping up to 2.5 mm., moderate ptosis from three to five mm., and marked ptosis over five mm., and grade 4 is a marked

TABLE 3*

RESULTS WITH ACQUIRED TRAUMATIC PTOSIS
(Eight Cases)

Type of Trauma	Amount of Resection	No. Cases	Excellent	Good		Poor	
				Slight Under Correction	Slight Over Correction	Considerable Undercorrection	Considerable Overcorrection
Blunt injury	12–15	2	0	0	0	0	2
Levator cut	6–10	6	4	1	0	1	0

* Results in ptosis following blunt injuries and in ptosis following perforating injuries with section of the levator attachment. The results are tabulated as in preceding tables.

TABLE 4*

DEGREE OF CONGENITAL PTOSIS VS. RESULT
(97 Cases)

Degree of Ptosis	No. Cases	Excellent Correction	Good		Poor	
			Slight Under Correction	Slight Over Correction	Considerable Undercorrection	Considerable Overcorrection
Mild 0–2.5 mm.	2	2 (100%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Moderate 3–5 mm.	48	48 (83%)	3 (6%)	2 (4%)	3 (6%)	0 (0%)
Marked 5+ mm.	37	22 (59%)	6 (16%)	0 (0%)	9 (24%)	0 (0%)
Marked ē levator action	10	0 (0%)	0 (0%)	0 (0%)	10 (100%)	0 (0%)

* Influence of degree of congenital ptosis upon result: Mild ptosis includes drooping up to 2.5 mm., moderate ptosis from 3.0 to 5.0 mm., and marked ptosis over 5.0 mm., and grade 4 is a marked ptosis with no levator action. As in previous tables, an "excellent" result is approximately within 1.0 mm. of the desired lid level and is symmetric with a good lid fold. A "good" result is within 2.0 to 2.5 mm. of the desired lid level (slight under or slight overcorrection). A "poor" result is 3.0 mm. or more under or over the desired lid level. The incidence of undercorrection appears to increase with more severe degrees of ptosis.

ptosis with no apparent levator action. In the moderate ptosis group, there were 83 percent excellent results, six percent slight undercorrections, and four percent slight overcorrections. Only six percent had a considerable undercorrection. In the 37 cases of the marked ptosis group, there were 59 percent excellent results, and 16 percent were slight undercorrections, 24 percent were considerably undercorrected. Thus, the incidence of undercorrection was appreciably higher with more marked degrees of ptosis. All of the 10 cases with marked ptosis and without any demonstrable levator action were considerably undercorrected. It should be mentioned, however, that some raising of the lid and some slight levator action or pseudolevator action was attained in some of these cases. In such cases of marked ptosis with no apparent levator action, the levator-frontalis sling operation may be preferred[8] (some levator or pseudolevator action is possible from effective shortening of the levator, the lid is raised to the desired position by attachment of the levator sling to the frontalis, and auxiliary movement is provided by the frontalis) or a frontalis operation or a superior rectus operation may be used. This avoids the disappointment of the frequent considerable undercorrections with levator resection alone, as seen in Table 4.

Table 5 shows the influence of levator action on the results in 97 cases of congenital ptosis. With fair to good levator action, the results were 82 percent excellent, seven percent slightly undercorrected, and three percent slightly overcorrected. Only eight percent were considerably undercorrected. With poor levator action evidenced by less than two mm. of levator movement, 26 percent were considerably undercorrected. However, 52 percent were excellent results and 22 percent were slight undercorrections. The persistence of a good percentage of successful results despite poor levator action confirms the dictum that levator resection is indicated as long as some apparent levator action is present. Again, with no levator action, 100 percent of the cases fell into the considerably undercorrected group.

Table 6 shows three patients or six lids with blepharophimosis and marked ptosis, and all of these cases had a considerable undercorrection. Table 6 also illustrates the re-

TABLE 5*

LEVATOR ACTION IN CONGENITAL PTOSIS VS. RESULT
(97 Cases)

Degree Levator Action	No. Cases	Excellent	Good		Poor	
			Slight Under Correction	Slight Over Correction	Considerable Under-correction	Considerable Over-correction
Fair to good	60	49 (82%)	4 (7%)	2 (3%)	5 (8%)	0 (0%)
Poor	27	14 (52%)	6 (22%)	0 (0%)	7 (26%)	0 (0%)
None	10	0 (0%)	0 (0%)	0 (0%)	10 (100%)	0 (0%)

* Influence of degree of levator action upon results: Poor levator action was evidenced by less than 2.0 mm. of levator movement. Results are classified as in previous tables. The incidence of undercorrection appears to increase with a lesser degree of levator action.

sults when jaw-winking of mild degree was associated with ptosis of moderate to marked degree. Three cases of moderate ptosis with slight jaw-winking all had satisfactory results with two classified as excellent, and one lid slightly undercorrected. The jaw-winking persisted in these cases, but was not marked enough to be a serious problem. It should be emphasized that only a slight amount of jaw-winking was present in these particular cases. In the one case of marked ptosis with marked jaw-winking, the ptosis still remained considerably undercorrected after operation. With marked jaw-winking levator resection methods are probably contraindicated.

Table 6 also shows eight cases with previously unsuccessful levator operations by various methods, which, when reoperated by the Iliff technique, had an excellent result in seven cases, and a slight undercorrection in one case. The levator resection varied from six to 12 mm. in these cases. Among these eight cases were three previous Iliff operations, three previous Blaskovics operations, and two previous Everbusch operations. Reoperation on these cases using the Iliff technique seemed easier with better hemostasis

TABLE 6*

RESULTS OF ILIFF TECHNIQUE ON SPECIAL PTOSIS PROBLEMS

Type	No. Cases	Excellent	Good		Poor	
			Slight Under Correction	Slight Over Correction	Considera-ble Under-correction	Considera-ble Over-correction
Congenital ptosis with blepharophimosis	6	0	0	0	6	0
Moderate ptosis with slight jaw-winking	3	2	1	0	0	0
Marked ptosis with marked jaw-winking	1	0	0	0	1	0
Congenital ptosis with previous unsuccessful levator resection	8	7	1	0	0	0

* The jaw-winking was of very slight degree associated with a moderate amount of ptosis (2nd row) or a marked amount of ptosis (3rd row). It should be emphasized that the Iliff technique was used on cases with minimal jaw winking only. The group which had a previous unsuccessful levator resection by various methods (last row) included three previous Iliff techniques, three previous Blaskovics techniques, and two previous Everbusch operations. Results are tabulated as in previous tables.

TABLE 7*

BILATERAL CONGENITAL PTOSIS OF UNEQUAL DEGREE
(One Side = Grade 2, Other Side = Grade 3)

No. Patients	Both Lids Excellent	Lid with Grade 3 Ptosis Undercorrected	
		Slight	Considerable
6	4	1	1

* Results in bilateral congenital ptosis of unequal degree: Four patients had an excellent result with both lids at a perfect or near perfect level. In two other bilateral ptosis cases, one case had the preoperatively more ptotic lid slightly undercorrected after operation, and the other case had the originally more ptotic lid moderately undercorrected after operation.

and a shorter operating time than would otherwise be expected with other levator resection techniques.

It is frequently stated that bilateral congenital ptosis of unequal degree is a problem because the lid with the more marked ptosis is frequently undercorrected. In six such patients after bilateral Iliff type procedures, four patients achieved a perfect or near perfect level with both lids (table 7). In one patient, the lid with the more marked ptosis still had a small, relative amount of ptosis remaining, and in one patient the lid with the most marked ptosis had a considerable relative amount of ptosis remaining.

SUMMARY

The results of 115 modified Blaskovics operations based on the Iliff technique have been analyzed. The percentage of good results with this modification compares favorably with previous reports of other techniques in the literature. There were no complications from the conjunctival resection. The resection of levator and conjunctiva together simplified the operation and perhaps made the levator resection more effective. Certain disadvantages were noted which seem inherent in all methods of levator resection, such as: postoperative lid lag which seemed more marked in cases with poor levator action, poor results in cases with no apparent levator action, and the susceptibility of acquired nontraumatic ptosis to overcorrection.

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REFERENCES

1. Iliff, C. E.: A simplified ptosis operation. Am. J. Ophth., **37**:529 (Apr.) 1954.
2. Berke, R. N.: Blepharoptosis. Arch. Ophth., **34**:434 (Nov.-Dec.) 1945.
3. ———: Congenital ptosis. Arch Ophth., **41**:188 (Feb.) 1949.
4. ———: Surgical correction of blepharoptosis. Am. J. Ophth., **36**:765 (June) 1953.
5. Johnson, C. C.: Blepharoptosis: A general consideration of surgical methods. Am. J. Ophth., **38**:129 (Aug.) 1954.
6. Cusick, P. L., and Sarrail, J. A.: Blepharoptosis: Modifications of Blaskovic's procedure, its indications, management of its complications. Kresge Eye Institute Bull., 2:38 (Nov.) 1950.
7. Schimek, R. A.: New techniques in ptosis surgery. Henry Ford Hosp. Bull., **2**:89, 1954.
8. ———: A new ptosis operation utilizing both levator and frontalis. Arch. Ophth., **54**:92 (July) 1955.
9. Verhoeff, F. H.: Personal communication.
10. Guyton, J. S.: Personal communication.
11. Berke, R. N., and Wadsworth, J. A. C.: Histology of levator muscle in congenital and acquired ptosis. Arch. Ophth., **53**:413 (Mar.) 1955.

PUPIL BLOCK IN APHAKIC EYES*

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Although pupillary block in aphakic eyes was recognized as early as 1865 by Bowman,[1] no systematic consideration of this subject appeared in the literature until the work of Chandler and Johnson.[2] Three cases are presented in order to emphasize the importance of this little-understood condition. In one case relief of pupil block was obtained before the development of angle block and increased intraocular pressure.

Chandler and Johnson described four types of eyes in which secondary glaucoma was due to pupillary block:

1. Aphakic eyes, with and without iridectomy, in which the pupil is blocked by a membrane, partly or wholly inflammatory.

2. Aphakic eyes, nearly always without iridectomy, in which the pupil is blocked by vitreous hernia, usually after discission of secondary cataract.

3. Eyes in which, after intracapsular extraction of the lens, the pupil is blocked by adhesion of iris to the intact hyaloid membrane.

4. Eyes with partial or complete subluxation of the lens backward, in which the pupil is blocked by herniation of vitreous through it.

Chandler[3] in discussing complications after cataract extraction, deleted the fourth type mentioned above and described four variations of pupil block in aphakic eyes. Two of these were included in the second type mentioned above and were separated into those following discission and those which occurred without discission.

The cases which will be presented here all followed round-pupil cataract extraction with two peripheral iridectomies in each instance. In no case was there any question of wound leak. No choroidal separation could be observed. Each case differed in its course, yet all had certain basic similarities. In each the hyaloid membrane remained intact. In each case both peripheral iridectomies became blocked, although this was not recognized at first. In each case the pupil could be dilated and constricted to some extent. In each case the anterior chamber formed normally after operation but became shallow later and in each it became deep following successful drainage of aqueous humor plus reestablishment of flow between the posterior and anterior chambers. In one case treatment was possible before the angle became blocked and thus before glaucoma had developed.

Case reports

Case 1

Mrs. M. B., aged 60 years, had an operation for extraction of an immature cataract on July 8, 1954. A McLean flap and three McLean sutures were used. An attempt at intracapsular extraction was unsuccessful and the capsule broke. The posterior cortex remained after the nucleus and most of the capsule were removed. Two peripheral iridectomies were made. About two weeks postoperatively the cortical material swelled and filled the pupillary space, the chamber became shallow, following which the tension rose to 48 mm. Hg (Schiøtz) on July 31st. The chamber remained shallow. On August 19, 1954, a peripheral corneal incision was made 1.5 mm. from the upper limbus and the lens material irrigated out. A reverse cyclodialysis was made in the upper temporal quadrant but the anterior chamber remained shallow and the tension became elevated, slightly at first, then up to 60 mm. Hg (Schiøtz). On October 1st a cyclodialysis was done through a scleral incision four mm. from the limbus in the lower temporal quadrant. At the same time a peripheral iridectomy was made in the upper nasal quadrant and air was injected into the anterior chamber before closure. Recovery was uneventful. The anterior chamber became normal in depth. The tension and corrected vision have been normal until the last examination in December, 1957.

In this case it was noticed during convalescence that the swelling of the remaining posterior cortical material occluded the pupil. It was not realized that the iridectomies were obstructed until after the cyclodialysis operation failed to deepen the anterior chamber in spite of the fact that cortical material had been washed out and the pupil no longer remained obstructed. An iridectomy-cyclodialysis per-

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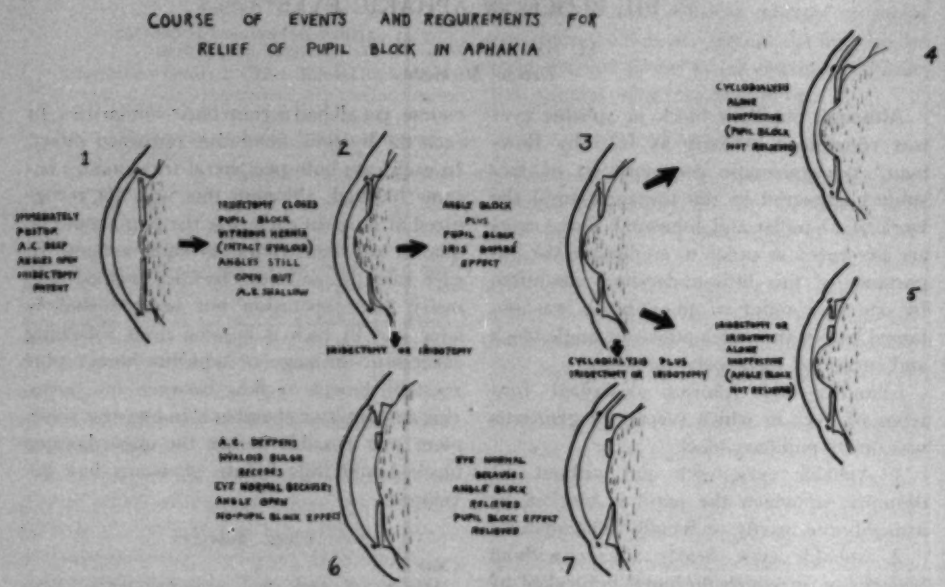

Fig. 1 (Sugar). Course of events and requirements for relief of pupil block in aphakia. In diagram 1 the normal relationships immediately postoperatively are shown. If the peripheral iris opening closes and mild inflammation produces adhesions between the hyaloid membrane and iris, resulting in pupil block, the vitreous bulges forward and the anterior chamber shallows as shown in diagram 2. If allowed to continue, the angles become blocked due to the iris bombé as shown in diagram 3. Diagram 4 shows the failure of cyclodialysis alone while diagram 5 shows failure of iridotomy or iridectomy alone. If a combination of both is used, relief of pupil block and angle block are obtained (diagram 7). In cases such as Case 3 in this paper, where iridotomy or iridectomy is done before the angle block occurs, the pupil block is relieved as shown in diagram 6.

mitted drainage through the obstructed angle and between the anterior and posterior chambers. The chamber deepened thereafter.

CASE 2

Mrs. B. B., aged 63 years, had had a round pupil intracapsular cataract extraction on December 12, 1957. A McLean flap and five corneoscleral sutures were used. Two iridectomies had been made in the far periphery of the iris. Recovery was uneventful. On January 28th the anterior chamber was noted to be shallow with the hyaloid membrane bulging forward. The pupil border was adherent to the hyaloid membrane in places but was generally free. No choroidal separation or wound leak was found. The tension was 40 mm. Hg (Schiøtz, 1955 scale). Atropine failed to improve the condition. Phospholine iodide similarly was ineffective. The chamber angle was blocked and the locations of the iridectomies could barely be seen in the periphery. A cyclodialysis was done on February 19th, under local anesthesia, in the inferior temporal quadrant but, although uncomplicated, the tension remained elevated. On February 20th a cyclodialysis was done at the 12-o'clock area just at the level of the insertion of the superior rectus muscle. Air was injected and a Wheeler knife iridotomy made in the temporal iris. The patient was seated on the operating table and taken back to her room in a wheelchair. The anterior chamber was deep the next day and has remained thus since. The tension has been soft. An open cyclodialysis cleft is present.

This case is important in showing the lack of value of cyclodialysis without producing a communication between the anterior and posterior chambers. When such a communication was established, the anterior chamber deepened and the vitreous bulge receded.

CASE 3

U. G. N., aged 50 years, had a right round-pupil, intracapsular cataract extraction with two peripheral iridectomies on April 24, 1957. A McLean flap and five corneoscleral sutures were used. His convalescence was uneventful and on June 11th the corrected vision was 20/20 with this eye. The anterior chamber was of normal depth. On October 3, 1957, shallowness of the anterior chamber was noted. The tension was 17 mm. Hg (Schiøtz, 1955 scale). No wound leak or choroidal separation was

present. Bulging of the intact hyaloid membrane was evident, nearly touching the cornea. The pupil border was free. The angle was shallow but open. The peripheral iridectomies were closed. Mydriasis and miosis had no effect on the anterior chamber depth. On October 19th a Wheeler knife incision was made temporally through the cornea and peripheral iris. The following day the anterior chamber was deep. The tension has remained normal since.

This case is the first described in which the late development of a shallow anterior chamber led to treatment for the relief of pupil block before angle block occurred.

Comments

One may summarize pupil block in aphakia as due to a combination of blocking of the pupil opening as well as absence or closure of all iridectomy or iridotomy openings between the anterior and posterior chambers. The pupil block is due to close contact or adhesion between the hyaloid membrane and the pupil border or the presence of an inflammatory membrane secluding the pupil. In some cases there is presumably complete posterior adhesion of the iris to the intact hyaloid membrane, even though the pupil border itself may be completely or partly free.

These requirements for pupil block in aphakia occur usually in round pupil extractions where the peripheral iridotomy or iridectomy openings are closed by inflammatory membranes or adherence to the surgical wound. Iridotomies certainly can more easily close. Iridectomy or iridotomy openings should not be made in the far periphery or, if they are, should be reasonably large. Two iridectomies are better than a single one. Although prolapse of iris predisposes to closure of iris openings, prolapse rarely occurs in these cases with good wound closure.

Late shallowness of the anterior chamber without evidence of wound leak or choroidal separation should lead one to suspect the presence of pupil block, even before any elevation of intraocular pressure. It should be particularly suspected with any elevation of tension. Chandler used a lower tension value of 22 mm. Hg in designating pupil block but it is more likely that pupil block can occur even in the presence of hypotony if there is marked suppression of aqueous formation. Swan[7] cites the example of marked iris bombé formation which is occasionally seen without glaucoma in the late stages of severe chronic uveitis with pupillary membrane formation. In these eyes aqueous formation is markedly reduced due to atrophic changes in the ciliary body. He states that it is probable that in the postoperative period after cataract extraction a similar suppression of aqueous formation occurs.

One may find an uneven depth of the anterior chamber in some cases when an inflammatory membrane stretches across part of the chamber and prevents the entire chamber from becoming shallow (Chandler[3]). Such an occurrence should arouse suspicion of pupil block.

The possibility of wound leak associated with loss of anterior chamber volume and choroidal separation must be considered in late emptying of the anterior chamber and was considered in the cases described, but was not found. In each case three to five corneoscleral sutures with a limbus based flap were used. The sutures included one preplaced McLean suture and two to four postplaced sutures of the same type. The anterior chamber was reformed at the first dressing in each case and remained so during the early postoperative period.

Biomicroscopy of the anterior segment always reveals herniation of the hyaloid-covered vitreous into the anterior chamber. In the normal vitreous of the child after discission free vitreous can produce block in the same manner. However, in the older adult it is unlikely that free vitreous herniating through a rent in the hyaloid membrane produces such block.

Reese[4] and Villasecca[5,6] discussed postoperative herniation of vitreous into the anterior chamber in cases without wound leak but considered it due to an increase in vitreous volume. Villasecca considered that an increase in vitreous pressure by coughing,

vomiting, or muscular effort could also produce this herniation. In addition, he listed dilatation of the pupil or broad iridectomy as factors in assisting the formation of vitreous hernia. Neither of these authors considered the presence or absence of communication between the anterior and posterior chambers through the iridectomy openings.

The question of mydriasis or miosis as a means of relieving pupil block and vitreous hernia in aphakic eyes has been answered both ways. Reese[4] advised pupillary dilatation with 10-percent phenylephrine, then constriction with isoflurophate. Kronfeld.[9] believes mydriatics to be advisable. If the pupil is completely free, such mydriasis should relieve pupil block. However, in cases such as those described herein, it is likely that adhesions between the posterior iris surface and hyaloid membrane, even if incomplete, make either miotics or mydriatics ineffective.

The treatment of pupil block may be summarized as follows:

If no increase in intraocular pressure is present (that is, the angle is still unblocked) iridotomy is all that is necessary to bypass the interference with communication between the anterior and posterior chambers (fig. 1, diagrams 2 and 6). In doing discission with a needle-knife in congenital cataracts, an iridotomy should be done with the knife just before withdrawing it from the eye.

If the intraocular pressure is elevated, both the angle block and pupil block must be relieved. A combination of cyclodialysis and iridotomy is therefore essential. In Figure 1, diagrams 3, 4, 5, and 7 show the failure of either cyclodialysis alone or iridectomy alone to relieve the condition. In diagram 7 the combination of cyclodialysis and iridotomy or iridectomy relieve both pupil block and angle block.

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References

1. Bowman, W.: On extraction of cataract by a traction-instrument with iridectomy with remarks on capsular obstructions and their treatment. Ophth. Hosp. Rep. London, **4**:332, 1863.
2. Chandler, P. A., and Johnson, C. C.: A neglected cause of secondary glaucoma in eyes in which the lens is absent or subluxated. Arch Ophth., **37**:740, 1947.
3. Chandler, P. A.: Complications after cataract extraction: Clinical aspects. Symposium: Cataract extraction. Tr. Am. Acad. Ophth., **58**:382, 1954.
4. Reese, A. B.: Herniation of the anterior hyaloid membrane following uncomplicated intracapsular cataract extraction. Tr. Am. Ophth. Soc., **46**:73, 1948.
5. Weisel, J., and Swan, K. C.: Mydriatic treatment of shallow anterior chamber after cataract extraction. Arch. Ophth., **58**:126, 1957.
6. Villasecca, A.: Correspondence. Arch. Ophth., **59**:150, 1958.
7. Swan, K. C.: Correspondence. Arch. Ophth., **59**:151, 1958.
8. Villasecca, A.: Late emptying of anterior chamber and choroidal detachment in cataract operations. Arch. Ophth., **52**:250, 1954.
9. Kronfeld, P. C.: Delayed restoration of the anterior chamber. Am. J. Ophth., **38**:453, 1954.

THE ETIOLOGY OF THE SO-CALLED A AND V SYNDROMES*

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The addition of the terms "A and V syndromes" to ophthalmic literature has been of value to the extent that attention has been focused on the straight-up and straight-down positions of gaze. The "A" refers to the fact that, in esotropia, the deviation is greater looking straight-up than straight-down, while in exotropia the deviation is greater looking down. The "V" refers to the fact that in esotropia the deviation is greater looking down while in exotropia it is greater looking up. These syndromes are found in cases that have a combined horizontal and vertical imbalance.

The incidence of the A and V syndromes makes them among the most common findings in squint cases. As an example, about 75 percent of the squint patients at the Illinois Eye and Ear Infirmary had a combined horizontal and vertical deviation and at least 50 percent of them showed an A or V syndrome.

In 1951[1] I was able to classify into four distinct groups the cases showing marked changes in the deviation of the eyes as the patients looked straight-up and straight-down. The classification was based on what the eyes did in vertical and lateral versions and also on characteristic cover measurements. Evidence, based on anatomy, on variations found with cover measurements, and on the results of surgery, was presented to show that the vertical deviations found in the four groups were secondary to the horizontal squint. Now let us see how the terms "A" and "V" syndromes have evolved from this classification.

In Group 1 cases, the complete descriptive terminology was "esotropia with bilateral elevation in adduction and a greater esotropia looking down than up." Substituting for the greater esotropia looking down than up we get "esotropia with bilaterial elevation in adduction and the V syndrome." Eliminating the "elevation in adduction," since most patients have this, we get "esotropia with the V syndrome, or V-esotropia." In the other three groups, by the same reasoning, V-exotropia is the same as Group III—exotropia with bilateral elevation in adduction and greater exotropia looking up than down; A-esotropia represents Group II—esotropia with bilateral depression in adduction and greater esotropia looking up than down; and A-exotropia is the same as Group IV—exotropia with bilateral depression in adduction and greater exotropia looking down than up.

An explanation of the remarkable changes seen in these positions is necessary. There is good evidence that the "A" and "V" syndromes can be produced by derangements in the vertical ocular muscles. Brown,[2] basing his ideas on the physiology of the vertical muscles, beautifully showed how they could produce the "A" and "V" syndromes. In the "A" syndrome, when looking up with the elevators contracting, the increased adduction of the eyes could be caused by overacting superior rectus muscles (whose secondary action is adduction) and by underacting inferior oblique muscles (whose secondary action is abduction). With the eyes looking down and the depressors contracting, the increased abduction could be due to overacting superior oblique muscles (secondary action abduction) and to underacting inferior rectus muscles (secondary action adduction).

In the "V" syndrome, when looking up with the elevators contracting, the increased abduction would be due to overacting inferior oblique muscles and underacting superior rectus mucles; when looking down with the depressors contracting, the increased adduction would be due to overaction of the in-

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ferior rectus muscles and underacting superior oblique muscles.

I am in complete agreement with his thesis that in primary vertical deviations the secondary horizontal deviations looking straight up and straight down are produced in this manner. But I believe there is also good evidence that the "A" and "V" syndromes can be found in cases where the primary pathology is in the horizontal muscles. This differentiation is not of theoretical interest only since the treatment in these cases depends on the etiology. The problem is how to differentiate between a primary vertical and a primary horizontal deviation in the "A" and "V" syndromes. For example, let us look at cases of esotropia with bilateral elevation in adduction. I believe, along with Dickey,[3] Swan,[4] and Scobee,[5] that in these cases the overaction of the inferior oblique muscles may be functional.

Now, what positions do the eyes assume when the patients look straight-up and straight-down in these cases of functional overaction of the inferior obliques? I have found that in most cases the "V" syndrome was present. Certainly it would be unlikely that the "V" syndrome in these patients would be due to a primary vertical muscle defect. It has been my experience that the "A" and "V" syndromes can result from dysfunction of the horizontal rectus muscles in those cases that fall into my four groups. I have presented evidence[6, 7] that in the "V" syndrome the greater esotropia down was due to overaction of the medical rectus muscles while the greater exotropia up was due to overaction of the lateral rectus muscles. In the "A" type of case the greater deviation down in exotropia was due to underaction of the medial rectus muscles and the greater deviation up in esotropia to underaction of the lateral rectus muscles.

It is the purpose of this paper to present further evidence that the "A" and "V" syndromes can be caused by dysfunctions of the horizontal rectus muscles.

Case reports

Case 1 (1-52)

An eight-year-old girl was seen at the infirmary on June 2, 1947, with a history of the eyes turning in since she was two years of age.

Refraction with atropine cycloplegia was: O.D., +3.0D. sph. ⊃ +0.5D. cyl. ax. 90°, 20/30; O.S., +2.75. sph. ⊃ +0.5D. cyl. ax. 180°, 20/30.

Preoperative examination (fig. 1) revealed about 20 degrees of esotropia with glasses and 25 degrees without. "V" esotropia was present with 30 degrees looking down and 10 to 15 degrees looking up. Bilateral elevation in adduction was easily seen.

Prism cover measurements were: c/c ET 24Δ; ET' 28Δ. s/c ET 40Δ; ET' 45Δ.

	ET 50	ET 39, RH 1	ET 40	
Right	ET 55	ET 45	ET 48	Left
	ET 40	ET 48	ET 45	

At surgery, on August 22, 1947, a four-mm. recession of the right medial rectus and a 10-mm. resection of the right lateral rectus muscle were done. One month after surgery (fig. 2) there was a large overcorrection of about 20 degrees of exotropia for distance. "V" exotropia was seen with 20 degrees looking up and straight eyes looking down. Cover measurements were: c/c XT 18Δ; XT' 12Δ s/c XT 12Δ, LH 1Δ; XT' 10Δ, LH 1Δ. Elevation of the left eye was now present in adduction while the right eye was level on gaze to the left. There was excess abduction of the right eye. Her glasses were removed and, after going without them for three months, she returned to the clinic with 10 to 15

Fig. 1 (Urist). Case 1. Preoperative appearance. (a) 25 degrees left esotropia without glasses. (b) and (c) Bilateral elevation in adduction. (d) About 10 to 15 degrees of esotropia looking up. (e) 30 degrees esotropia looking down.

Fig. 2 (Urist). Case 1. One month postoperatively after recession of the right medial rectus and resection of the right lateral rectus. (a) 20 degrees of right exotropia for distance. (b) Elevation of the left eye in adduction with increased abduction of the right eye. (c) Eyes level on lateral gaze to the left. (d) About 20 degrees left exotropia looking up. (e) Straight down.

degrees of esotropia. A reduced plus correction was prescribed which kept the eyes straight. When she was again seen in March, 1953, cover measurements were: c/c XT 5Δ, RH 1Δ; ET′ 8Δ, RH 1Δ. s/c ET 16Δ, RH 1Δ; ET′ 16Δ.

	XT 14	ET 8, RH 2	ET 4, RH 3	
Right	XT 2	ET′ 16	ET 3, RH 1	Left
	XT 12, RH 3	XT 5, RH 1	XT 8	

Second grade fusion was present at 0.

The glasses were worn constantly until March, 1956, when she returned to the clinic. Now a radical change was seen to have taken place (fig. 3). For near with glasses, 15 degrees of exotropia was present and without glasses, about five degrees. "A" exotropia was present with 25 degrees looking down and straight eyes looking up. Clinically the excess abduction of the right eye had improved but now increased limitation of the right eye in adduction was seen.

Cover measurements were: c/c XT 5Δ; XT′ 15Δ. s/c ET 7Δ, RH 2Δ; XT′ 5Δ.

	ET 7, RH 4	ET 4, RH 3	XT 4	
Right	ET 10, RH 2	XT′ 5	XT 7, RH 2	Left
	ET 5, RH 2	XT 3	XT 10	

Comment

In this case the prism cover measurements explained how the changes from "V" esotropia to "V" exotropia to "A" exotropia took place. Following surgery directed at correcting the esotropia, overaction of the right resected lateral rectus was produced. This caused excess abduction of the right eye producing a greater exotropia for distance and on looking up which was verified by the prism cover measurements, taken in 1953, that showed the greatest exotropia to be up and to the right, which is in the sphere of influence of the right lateral rectus muscle.[8]

With the wearing of plus lenses, through the years a weakness of convergence developed and the "A" exotropia was produced. Underaction of the recessed right medial rectus had become pronounced, as

Fig. 3 (Urist). Case 1. Nine years postoperatively. (a) 10 to 15 degrees right exotropia with glasses. (b) Eyes level on gaze to the right. (c) Limitation of the right eye in adduction. (d) Straight looking up. (e) 25 degrees right exotropia looking down.

Fig. 4 (Urist). Case 2. Preoperatively. (a) 30 degrees right esotropia with glasses. (b) Limitation of the left eye looking up and to the right. (c) Limitation of the right eye looking up and to the left. (d) About 40 degrees right esotropia looking up. (e) About 15 to 20 degrees left esotropia looking down.

evidenced by the greater exotropia measurements down and to the left, which is in the sphere of influence of the right medial rectus muscle.[8] In this case then "V" exotropia was caused by an overaction of the right lateral rectus and the "A" exotropia by weakness of convergence and underaction of the right medial rectus muscle.

CASE 2 (1-247)

A five-year-old girl was seen at the infirmary in July, 1947, with a history of her eyes turning in since three months of age.

Refraction with atropine cycoplegia was: O.D., +5.0D. sph., 20/50; O.S., +5.0D. sph. 20/50.

Preoperative examination (fig. 4) revealed 30 degrees of esotropia with glasses. "A" esotropia was present with 40 degrees looking up and 15 to 20 degrees looking lown. Bilateral depression in adducation was present, with limitation of the adducted eye in upward gaze (apparent underaction of the inferior oblique muscles).

At surgery, on August 8, 1947, bilateral 10-mm. resections of the lateral rectus muscles and a four-mm. recession of the left medial rectus muscle were done. Postoperative examination on July 3, 1957 (fig. 5), 10 years after operation, revealed the eyes to be straight with glasses and about 10 degrees convergent without. "V" accommodative esotropia was present with 20 degrees looking down and straight eyes looking up. Bilateral elevation in adduction was now seen (apparent overaction of the inferior obliques). She had been wearing the following correction: O.D., +5.5D. sph. 20/20; O.S., +6.0D. sph., 20/30.

Prism cover measurements at this time were: c/c ET 26Δ, RH 1Δ; ET' 30Δ. s/c ET 44Δ; ET' 43Δ. The convergence nearpoint was 40 mm.

	ET 26, LH 3	ET 40	ET 46	
Right	ET 30, LH 1	ET 43	ET 58	Left
	ET 42, LH 2	ET 48	ET 64	

COMMENT

The improvement in the action of the lateral rectus muscles following the bilateral resections resulted in the disappearance of the greater esotropia on looking up along with the underaction of the inferior obliques. Therefore the original "A" esotropia with functional underaction of the inferior obliques most likely was caused by a con-

Fig. 5 (Urist). Case 2. Ten years following bilateral resection of left lateral rectus and recession of left medial rectus muscles. (a) Straight with glasses. (b) Straight looking up. (c) About 20 degrees right esotropia looking down. (d) and (e) Bilateral elevation in adduction.

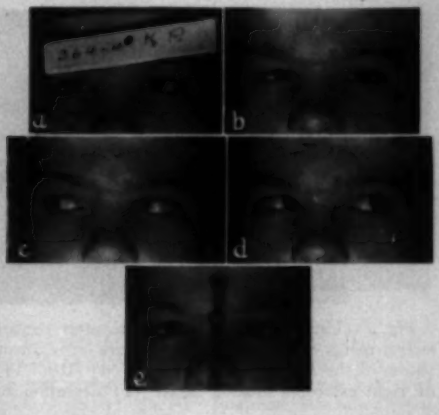

Fig. 6 (Urist). Case 3. Preoperatively. (a) 20 degrees of left esotropia. (b) 20 degrees of right esotropia. (c) and (d) Bilateral elevation in adduction. (e) Convergence near-point 40 mm.

genital underaction of the lateral rectus muscles.

Following the improvement of the "A" esotropia, after many years a new type of squint developed, namely, accommodative "V" esotropia. It was accommodative in type since the wearing of the strong plus lenses kept the eyes straight. Without glasses a convergence excess type of esotropia was present with overaction of the medial rectus muscles especially the right (unoperated) muscle, demonstrated by the greatest esotropia being down and to the left, the sphere of influence of the right medial rectus muscle.[8]

With the deveolpment of convergence excess and overaction of the medial rectus muscles the apparent underaction of the inferior obliques changed to bilateral overaction of the inferior obliques. In this case then the "A" esotropia with secondary underaction of the inferior oblique muscles was due to underaction of the lateral rectus muscles and the "V" esotropia with secondary overaction of the inferior oblique muscles was due to overaction of the medial rectus muscles.

CASE 3 (1-99)

A six-year-old girl was seen at the infirmary on May 8, 1948, with a history of the left eye turning in since seven months of age.

Refraction with atropine cycloplegia was: O.D. +5.75D. sph. 20/30; O.S., +2.5D. sph. finger counting at four feet.

About 20 degrees of left esotropia with eccentric fixation was present. After complete occlusion of the right eye for six months, vision in the left eye improved to 20/30.

Preoperative examination (fig. 6) revealed about 20 degrees of esotropia with and without glasses and bilateral elevation in adduction. The convergence nearpoint was 40 mm. and with the delayed cover test alternating sursumduction, excycloduction, and occlusion nystagmus were present.

Prism cover measurements were: s/c ET 56Δ, LH 24Δ; ET′ 80Δ, LH′ 6Δ.

At surgery, on January 21, 1949, a five-mm. recession of the left medial rectus and an eight-mm. resection of the left lateral rectus muscle were done. Postoperative examination, on December 20, 1950, two years after surgery (fig. 7), showed the eyes to be slightly divergent with glasses and five to 10 degrees convergent without glasses. The "V" syndrome was present with about 10 degrees of exotropia looking up and about 10 degrees of esotropia looking down. Bilateral elevation in adduction was still present.

Prism cover measurements were: c/c ET 29Δ, RH 8Δ; ET′ 25Δ, RH′ 8Δ, s/c ET 40Δ, RH 4Δ; ET′30Δ, RH 3Δ.

	ET 12, LH 12	ET 12	ET 23, RH 4	
Right	ET 14, LH 6	ET 30, RH 3	ET 28	Left
	ET 16, LH 5	ET 22	ET 32	

When the patient returned in March, 1956, the picture had changed markedly (fig. 8). Now there was 20 degrees of exotropia for distance and 15 degrees for near. "A" exotropia, with 20 degrees looking down and 10 degrees looking up, was present. Marked depression of the left eye in adduction was now seen. While the eyes were level on gaze to the left, alternating sursumduction, excycloduction, and occlusion nystagmus were still present. The convergence near-point was remote with bilateral limitation in adduction, greater in the left eye.

Cover measurements were: E 6Δ, RH 16Δ; RH′ 12Δ.

Fig. 7 (Urist). Case 3. Two years after recession of the left medial rectus and resection of the left lateral rectus muscles. (a) Five degrees left exotropia with glasses. (b) Straight without glasses. (c) Elevation of the right eye in adduction. (d) About 10 degrees of exotropia looking up. (e) About 10 degrees of right esotropia looking down. (f) Elevation and limitation of the left eye in adduction.

	XT 3	XT 4	LH 2	
Right	XT 16, RH 14	RH 12	LH 2	Left
	XT 10, RH 16	XT 10, RH 6	LH 4	

Comment

Following surgery a small "V" esotropia remained which was corrected by glasses. Bilateral elevation in adduction was still present with typical cover measurements in that the greatest left hypertropia was in dextro-elevation and the greatest right hypertropia was in levo-elevation. The cover measurements also showed that the characteristic effect of a left sided recession and resection was greatest on gaze to the right.[8]

With glasses, the eyes diverged for distance at times. But, since the cover measurements showed such a large esotropia, the glasses were not reduced. Along with the wearing of the full atropine correction, over the years, weakness of convergence developed, manifested by a remote convergence near-point and increased underaction of the medial rectus muscles as shown by bilateral limitation in adduction, greater on the left. This was confirmed by cover measurements in that the greatest exotropia was down and to the right, the sphere of influence of the left medial rectus muscle. As the underaction of the medial rectus muscles developed there was a shift from the "V" esotropia to "A" exotropia. Along with this the apparent overaction of the left inferior oblique muscle changed to an apparent underaction as the underaction of the left medial rectus muscle increased.

Fig. 8 (Urist). Case 3. Six years postoperatively. (a) About 20 degrees of exotropia for distance. (b) 10 to 15 degrees of left exotropia for near. (c) About five degrees of exotropia looking up. (d) Depression and limitation of the left eye in adduction. (e) Eyes level on gaze to the left with limitation in adduction of the right eye. (f) About 15 to 20 degrees of left exotropia looking down.

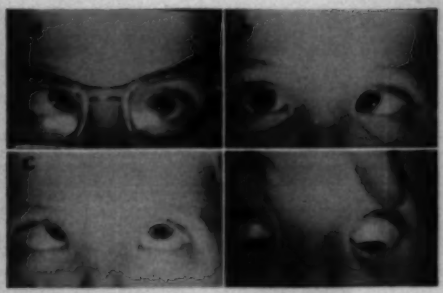

Fig. 9 (Urist). Case 4. Preoperatively. (a) 30 degrees of right esotropia with glasses. (b) 30 degrees of left esotropia without glasses. (c) 35 degrees of right esotropia looking up. (d) Five to 10 degrees of esotropia looking down.

CASE 4 (4-197)

A seven-year-old boy was seen at the infirmary on July 25, 1955, with a history of his eyes turning in since three months of age.

Refraction with atropine cycloplegia was: O.D., +2.75D. sph. ⊃ +0.75 cyl. ax. 80°, 20/30; O.S., +2.5D. sph., 20/30.

Preoperative examination (fig. 9) revealed 30 degrees of esotropia with and without glasses. "A" esotropia was present with 35 degrees looking up and only five degrees looking down. At surgery, on February 8, 1956, bilateral four-mm. recessions of the medial rectus muscles were done.

Postoperative examination, seven months after surgery (fig. 10), revealed the presence of 10 to 15 degrees of exotropia with and without glasses. "A" exotropia was now present with 20 degrees looking down and five to 10 degrees looking up. The eyes appeared level in lateral gaze but cover measurements were characteristic of bilateral depression in adduction. The convergence near-point was 30 mm.

Prism cover measurements were: c/c ET 18Δ, RH 6Δ; XT′ 20Δ, RH 10Δ. s/c ET 18Δ, LH 6Δ; XT′ 20Δ, LH 4Δ.

	XT 22	XT 12	XT 10	
Right	XT 34, RH 4	XT 20, RH 6, LH 4	XT 16	Left
	XT 40, RH 14	XT 40, RH 12	XT 24, LH 9	

The plus lenses were replaced with −1.0D. sph. in each eye. When seen on June 9, 1957 (fig. 11), he had about five degrees of esotropia with and without glasses. The "V" syndrome was now present with a slight esotropia looking down and five to 10 degrees of exotropia looking up. Along with this, bilateral elevation in adduction was present.

Cover measurements were: c/c ET 26Δ; ET′ 32Δ, RH 6Δ. s/c ET 20Δ, LH 4Δ; ET′ 10Δ, LH 6Δ.

	ET 3, LH 2	ET 5	ET 4, RH 8	
Right	ET 4, LH 4	ET 10, LH 5, RH 5	ET 10, RH 4	Left
	ET 8	ET 10	ET 8, RH 2	

COMMENT

I have found that "A" esotropia cases can be caused by a congenital underaction of the lateral rectus muscles.[9] A strengthening procedure directed primarily at the lateral rectus muscles, along with a weakening procedure to the medial rectus muscles if necessary, had given the best results.[10] In this case a small bilateral recession of the medial rectus muscles was done for a large esotropia of 30 to 35 degrees, yet it resulted in an unex-

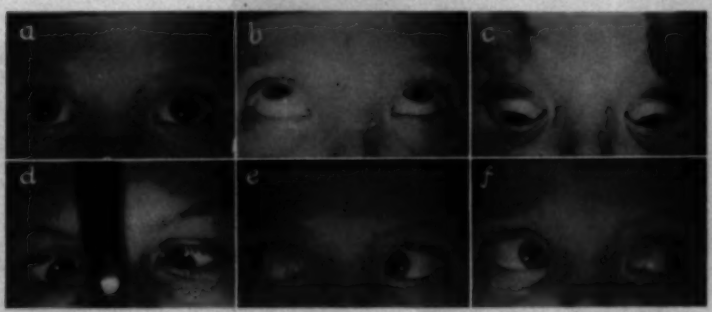

Fig. 10 (Urist). Case 4. Seven months after bilateral recessions of the medial rectus muscles. (a) 10 degrees of left exotropia. (b) Five to 10 degrees of right exotropia looking up. (c) 20 degrees of exotropia looking down. (d) Convergence near-point 30 mm. (e) and (f) Eyes level on lateral gaze.

Fig. 11 (Urist). Case 4. Postoperatively; 16 months after wearing —1.0D. sph. (a) Five degrees of left esotropia with glasses. (b) Five degrees of left esotropia without glasses. (c) About 10 degrees of right exotropia looking up. (d) Straight looking down. (e) and (f) Bilateral elevation in adduction.

pected overcorrection, namely, an "A" type exotropia. The muscles were not over-recessed and did not slip since a very good convergence near-point was present. The reason for the postoperative divergence was that only 10 degrees of esotropia were present looking down before surgery and, since the medial rectus muscles have their greatest effect in the down position, following their underaction an exotropia developed, greatest in this position.

The underaction of the medial rectus muscles also produced cover measurements that were diagnostic for bilateral depression in adduction even though it could not be seen on versions. The greatest right hypertropia was in dextro-depression and the greatest left hypertropia in levo-depression. The dynamic change in the medial rectus muscles from underaction to overaction, following the change in lenses, changed the "A" type exotropia with bilateral depression in adduction into a "V" type esotropia with bilateral elevation in adduction. This was confirmed with cover measurements since now the greatest right hypertropia had changed from dextro-depression to levo-elevation and the greatest left hypertropia from levo-depression to dextro-elevation.

CASE 5 (1-124)

A five-year-old boy was first seen at the infirmary on February 4, 1948, with a history of the eyes being crossed since birth.

Refraction with atropine cycloplegia was: O.D., +2.0D. sph., 20/50; O.S., +2.0D. sph., 20/50.

At preoperative examination (fig. 12) with the right eye fixing there was 35 degrees of esotropia,

Fig. 12 (Urist). Case 5. Preoperatively. (a) 35 degrees of left esotropia with a left hypertropia for near. (b) 35 degrees of right esotropia with a right hypertropia for near. (c) 35 degrees of left esotropia with a left hypertropia for distance. (d) Elevation of the left eye in adduction. (e) Elevation of the right eye in adduction. (f) Convergence near-point is 20 mm. (g) 30 degrees of left esotropia looking up. (h) 45 degrees of right esotropia looking down.

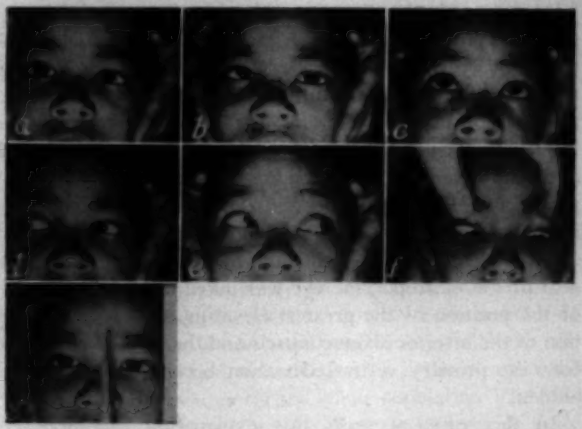

Fig. 13 (Urist). Case 5. Postoperatively—two months following recession of the left medial rectus and resection of the left lateral rectus muscles. (a) 20 degrees of left esotropia for near, no hypertropia (compare with fig. 12-a). (b) 20 degrees of right esotropia with a right hypertropia for near. (c) 10 to 15 degrees of left esotropia looking up. (d) Limitation of the left eye in adduction with slight elevation. (e) Marked elevation of the right eye in adduction. (f) 10 to 15 degrees of right esotropia looking down. (g) Convergence near-point is 90 mm.

10 degrees of hypertropia, and elevation of the left eye in adduction. With the left eye fixing there was 35 degrees of esotropia, 10 degrees of hypertropia, and elevation of the right eye in adduction. "V" estropia with 30 degrees looking up and 45 degrees looking down was present.

At surgery, on April 15, 1948, a six-mm. recession of the left medial rectus and nine-mm. resection of the left lateral rectus muscle were done. Postoperative examination (fig. 13), two months after surgery, revealed with the right eye fixing 20 degrees of esotropia with no hypertropia and no elevation in adduction of the left eye. With the left eye fixing there was 35 degrees of esotropia, 10 degrees of hypertropia, and elevation of the right eye in adduction. "V" esotropia was present with a little more esotropia down than up.

Cover measurements were: c/c ET 48Δ, RH 8Δ, ET′ 46Δ, RH 4Δ. s/c ET 50Δ, RH 2Δ, ET′ 48Δ, RH 2Δ.

	ET 28	ET 40, RH 5	ET 38, RH 9	
Right	ET 47	ET 48, RH 2	ET 52, RH 8	Left
	ET 45	ET 45, RH 4	ET 53, RH 5	

At reoperation, on July 15, 1948, a four-mm. recession of the right medial rectus mucle and an eight-mm. resection of the right lateral rectus muscle were done. Postoperative examination, on March 14, 1951, three years after surgery, revealed (fig. 14) the eyes straight for distance and most of the time for near. A slight exotropia could be present at times for near with a slight esotropia for distance. There was marked bilateral depression in adduction. The "A" syndrome was now present in that the eyes were straight looking up and 20 degrees divergent looking down.

Cover measurements were: ET 12Δ, LH 2Δ; E′ 6Δ, RH 6Δ.

	XT 5, RH 7	RH 5	XT 3	
Right	RH 10	E′6, RH 6, LH 3	XT 6	Left
	XT 16, RH 20	XT 7, RH 4	XT 6, LH 5	

Comment

Before surgery the nonfixing eye turned in 35 degrees and elevated 10 degrees (fig.

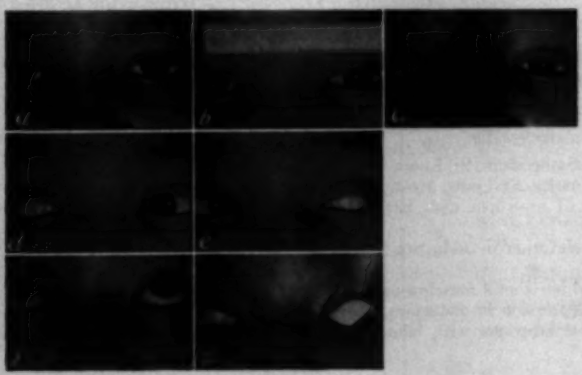

Fig. 14 (Urist). Case 5. Three years following second operation—recession of the right medial rectus and resection of the right lateral rectus. (a) Slight right esotropia for distance. (b) Straight for near. (c) Convergence near-point is 110 mm. (d) and (e) Bilateral depression and limitation in adduction. (f) Straight looking up. (g) 25 degrees of left exotropia looking down.

12). Bilateral elevation in adduction was present. After surgery to the left eye, when the right eye fixed, 20 degrees of left esotropia was present with no hypertropia and no elevation in adduction (fig. 13). Assuming the overaction of the inferior oblique was a primary vertical dysfunction, the disappearance of the hypertropia and elevation in adduction might be explained as follows: After surgery, with straightening of a great deal of the esotropia, the eye was taken out of the position of the greatest elevating action of the inferior oblique muscle and therefore the primary vertical deviation became hidden.

In this case, at least, this explanation would be unlikely because (1) when the right eye fixed in extreme abduction, the left eye was adducted sufficiently to place it in the field of action of the inferior oblique muscle and yet it did not elevate; (2) however, when the left eye fixed, the right (unoperated) eye still had 10 degrees of hypertropia and marked elevation in adduction (fig. 13); (3) then, when the horizontal muscles of the right eye were operated, the elevation of the right eye in adduction disappeared. This sequence of events would be proof that the bilateral elevation in adduction was secondary to a dysfunction of the horizontal rectus muscles.

After the final surgery underaction of the medial rectus muscles developed as shown by the limitation in adduction and the poor convergence nearpoint (fig. 14). The greatest underaction was of the left medial rectus muscle as shown by cover measurements in which the greatest exotropia was down and to the right. This was consistent since it had been recessed six mm. while the right medial rectus muscle was only recessed four mm. A new type of squint, "A" exotropia, with characteristic secondary vertical deviations, namely, bilateral depression in adduction, was produced. With cover measurements the right hypertropia changed from being greatest in levo-elevation to dextro-depression and the left hypertropia was greatest in levo-depression.

Summary

1. The "V" and "A" syndromes can be produced by dysfunction of the horizontal rectus muscles.

2. In the "V" syndrome, with apparent overaction of the inferior oblique muscles, the primary dysfunction was overaction of the horizontal muscles, the medial recti in esotropia and the lateral recti in exotropia.

3. In the "A" syndrome with apparent underaction of the inferior oblique muscles, the primary dysfunction was underaction of the horizontal muscles, the lateral recti in esotropia and medial recti in exotropia.

4. Evidence for the above statements was based on the results of known changes in action of the horizontal muscles which produced a change from "A" to "V" syndrome or vice-versa in the same patient.

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References

1. Urist, M. J.: Horizontal squint with secondary vertical deviations. Arch. Ophth., **46**:245, 1951.
2. Brown, H. W.: Symposium strabismus: Vertical deviations. Tr. Am. Acad. Ophth., March-April, 1953, p. 157.
3. Dickey, C. A.: Symposium strabismus: Surgery in treatment of strabismus. Tr. Am. Acad. Ophth., March-April, 1953, p. 174.
4. Swan, K.: Strabismus Ophthalmic Symposium. St. Louis, Mosby, 1950, p. 458.
5. Scobee, R. G.: The Oculorotary Muscles. St. Louis, Mosby, 1952, p. 380.
6. Urist, M. J.: Surgical treatment of esotropia with bilateral elevation in adduction. Arch. Ophth., **47**:270, 1952.
7. ———: Exotropia with bilateral elevation in adduction: Part II. Surgery. Am. J. Ophth., **38**:178, 1954.
8. ———: The effect of asymmetrical horizontal muscle surgery. Arch. Ophth., **59**:247, 1958.
9. ———: Esotropia with bilateral depression in adduction. Arch. Ophth., **55**:509, 1956.
10. ———: The surgical treatment of esotropia with bilateral depression in adduction. Arch. Ophth., **55**:643, 1956.

ARE AQUEOUS HUMOR DYNAMICS INFLUENCED BY AGING? II*

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In a previous paper[1] data were presented showing intraocular pressure (P), facility of outflow (C), and rate of outflow (F) of aqueous humor at four different age levels: 10-29, 30-49, 50-69, and 70-92. Statistical treatment of these data combined with opthalmologic considerations led to the conclusion that the average intraocular pressure, the average resistance to outflow, and the average rate of outflow do not change significantly with increasing age.

The average or arithmetic mean (M) of such data is the value about which the data tend to cluster. In addition to the average, collections of such data are characterized by another parameter: the standard deviation(s). This parameter, or its square, the variance, measures the spread of the data, or the closeness with which they cluster about the average. In this paper we give the results of further statistical treatment of the data presented in the previous paper,[1] this time considering primarily the information obtained from the standard deviations.

We shall use the word "normal" with two different meanings. In the statistical sense, a normal distribution is a collection of data such as intraocular pressures, P, which are distributed according to the equation:

$$y = \frac{N}{\sigma\sqrt{2\pi}} e^{-1/2[P-\mu/\sigma]}$$

The frequency of occurrence of a given pressure is denoted by y, N is the number of eyes in the population considered, σ is the standard deviation, and μ is the mean of the pressures for the whole population. The estimates of σ and μ obtained from a finite sample of eyes are designated, respectively, as s and M. In the ophthalmologic sense, the use of the word "normal" is illustrated by Friedenwald's definition of normal (or normative) intraocular pressure as that pressure which is compatible with continued health and function of the eye (quoted in reference[2]). In the following, the intended meaning of "normal" should be clear from the context.

METHOD

The selection of the subjects and the experimental procedures used have already been discussed.[1,3] The basic data were presented.[1]

For this paper, statistical treatment of the data was done in three steps. First, each distribution (of P, C, and F for the age groups 10-29, 30-49, 50-69, and 70-92) was tested for normality by calculating cumulative percentages from cumulative observed frequencies and then plotting the results on probability paper. A straight line is obtained if the distribution is normal, whereas a curved plot implies a non-normal distribution.

Second, results of the F-tests described[1] were considered further.

Third, normal curves were fitted to those distributions which were shown by the plots on probability paper to be normal. The fitting procedure is described.[4]

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As previously,[1] the data were grouped not only for the ages 10-29, 30-49, 50-69, 70-92, but also for the ages 10-49 and 50-92.

The deductions made from the statistical treatment were then considered in connection with the data of Alimuddin[5] and with the frequency data obtained from several glaucoma surveys.

Results

Intraocular pressure

Results of the test for normality for the largest group (ages 50-69, 225 eyes) are shown in Table 1 and Figure 1. The straight line relationship is an indication that the data are normally distributed. Similar results were found for the other age groups, 10-29, 30-49, and 70-92.

TABLE 1

Tests for normality, age group 50–69 years

P	Frequency	Cumulative Frequency	Cumulative (percent)
12.0	0	0	0.00
14.0	3	3	1.33
16.0	19	22	9.78
18.0	25	47	20.89
20.0	55	102	45.33
22.0	40	142	63.11
24.0	38	180	80.00
26.0	35	215	95.56
28.0	10	225	100.00
C	**Frequency**	**Cumulative Frequency**	**Cumulative (percent)**
0.120	12	12	5.33
0.180	38	50	22.22
0.240	59	109	48.44
0.300	57	166	73.78
0.360	31	197	87.56
0.420	12	209	92.89
0.480	10	219	97.33
0.540	3	222	98.67
0.600	1	223	99.11
0.660	1	224	99.56
0.720	1	225	100.00
F	**Frequency**	**Cumulative Frequency**	**Cumulative (percent)**
1.95	7	7	3.11
3.25	44	51	22.67
4.55	75	126	56.00
5.85	52	178	79.11
7.15	19	197	87.56
8.45	14	211	93.78
9.75	8	219	97.33
11.05	6	225	100.00

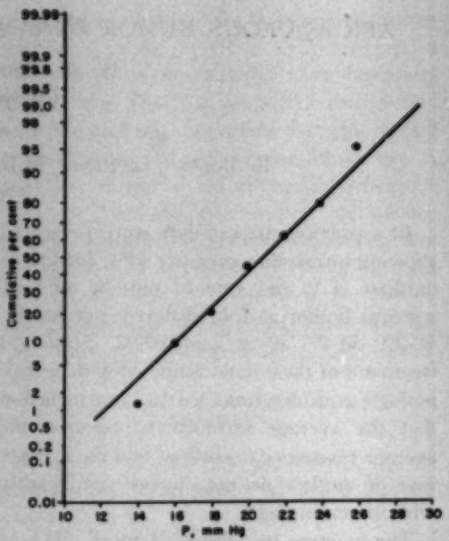

Fig. 1 (Spurgeon, et al.). Test for normality, age group 50-69 years.

Figure 2 shows how the spreads of intraocular pressures increase with increasing age. Application of the F-test (table 7, ref. 1) shows that the spread of intraocular pressures is not significantly different when age groups that are closest together are compared: 10-29/30-49, 10-29/50-69, 30-49/50-69 and 50-69/70-92. For age groups that are farther apart the differences in spread are significant. The spread is greater for the 70-92 group than for the 30-49 group (significantly different at the five-percent level). It is still greater than for the 10-29 group (significantly different at the one-percent level).

Similarly, while the average intraocular pressure is not significantly different for the 50-92 group than for the 10-49 group, the spread of pressures is significantly greater for the older group. In other words, while the older subjects as a group had the same average pressure as the younger subjects, there were more older people whose pressures deviated widely from the mean.

In order to illustrate this further and to make possible some comparisons with data

from other sources, normal curves were fitted to the intraocular pressure data. Some question might be raised as to whether this procedure is justifiable, since we are dealing with distributions truncated above P = 30 mm. Hg. It is known from other data, however, that the truncated portions of the curves are quite small, consequently the estimate of the means and the standard deviations cannot be very different from those that would be obtained if the truncated portions were considered, or if the more complicated iteration procedures were used to obtain the estimates. It is therefore considered that the estimates M and s obtained directly from the data are very good approximations to the values that would have been obtained by the more complicated procedures.

The normal curves obtained for the four age groups are shown in Figure 3. The data were all adjusted to the total of the age group (50-69 years) having the largest number of eyes (225). The shaded areas give the expected frequencies of pressures above 30 mm. Hg in a normal population. These areas were calculated with the aid of normal probability tables, giving the percentages shown in Figure 3 and Table 2. In Figure 4 these areas are magnified to show better the increases with age.

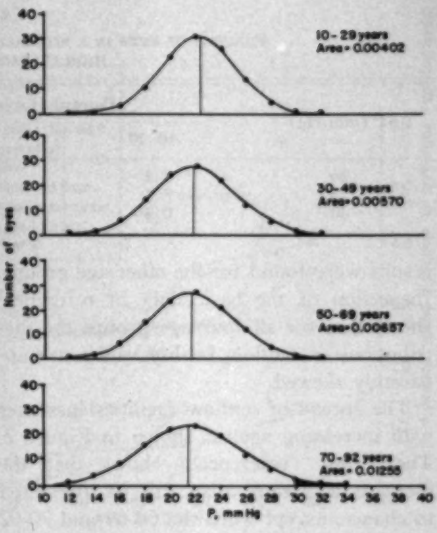

Fig. 3 (Spurgeon, et al.). Normal distribution curves fitted to the intraocular pressure data. Vertical lines are arithmetic means. All data are reduced to the same total number of eyes (225). The progressive flattening of the curves with increasing age illustrates the concurrent increase of spread. (Note heavier black area at right end of each curve.)

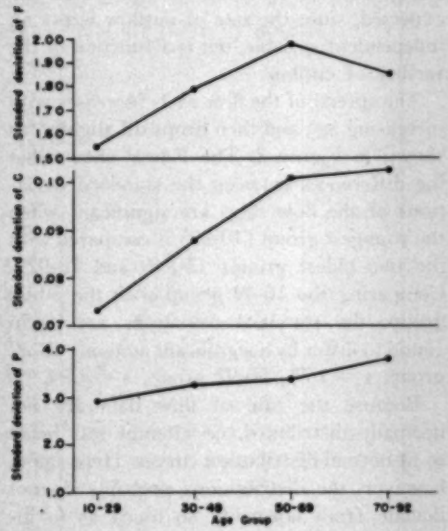

Fig. 2 (Spurgeon, et al.). Effect of age on spread of tonographic values. In the older age groups, in general, there are more individuals whose tonographic values differ widely from the average values.

In Figure 5 we have plotted in solid lines the percent of eyes having intraocular pressures greater than 28 mm. and 30 mm. These percentages were calculated from the normal curves fitted to our data. For the plots in Figure 5, 20 years was taken as the average age for the 10-29 group; 40 years for the 30-49 group; 60 years for the 50-69 group; and 80 years for the 70-92 group. The graphs so obtained, therefore, are not quantitatively exact, but they do serve to show the trend.

Facility of Outflow

Results of the test for normality for the largest group (ages 50-69, 225 eyes) are shown in Figure 6. It is apparent that the data are not normally distributed. Similar

TABLE 2

PERCENT OF EYES IN A NORMALLY DISTRIBUTED POPULATION HAVING HIGH INTRAOCULAR PRESSURE

P (mm.Hg)	Percent Having Equal or Greater Intraocular Pressure			
	10–29	30–49	50–69	70–92
26	8.4	7.8	7.8	8.7
28	2.4	2.6	2.7	3.6
30	0.40	0.57	0.66	1.3

results were found for the other age groups. Inspection of the basic data in reference[1] shows that for all four age groups the distributions of outflow facility values are apparently skewed.

The spread of outflow facilities increases with increasing age, as shown in Figure 2. The F-test (reference[1]) shows that the increase is greater than can be attributed to chance, except when the 50-69 and 70-92 groups are compared. When the 10-49 and 50-92 groups are compared as was done in[1] the spreads are very different, being significantly greater (one-percent level) for the older group: 10-49 group, $s = 0.0851$; 50-92 group, $s = 0.102$.

Because the facility of outflow data are not normally distributed, no attempt was made to fit normal distribution curves. It should be pointed out, however, that the distributions probably do not deviate from normality so much as to invalidate the use of the t- and F-tests.

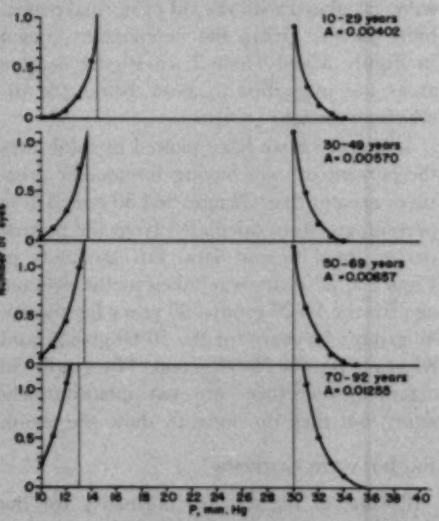

Fig. 4 (Spurgeon, et al.). Areas under the normal curves of Figure 3, magnified to show the increases with age of the number of eyes having P greater than 30 and P less than 13 to 14 mm.

RATES OF FLOW

Results of the test for normality for the largest group are shown in Figure 7. Here again the data are not normally distributed. Similar results were found for the other age groups. Inspection of the basic data in reference[1] shows that for all four age groups the distributions of flow rate values are apparently skewed. These results are not unexpected, since the rate of outflow is not an independent variable, but is a function of the facility of outflow.

The spread of the flow rates increases with increasing age and then drops off slightly, as shown in Figure 2. The F-test[1] shows that the differences between the standard deviations of the flow rates are significant when the youngest group (10-29) is compared with the two oldest groups (50-69 and 70-92). Comparing the 10-49 group with the 50-92 group, the standard deviations are again found to differ by a significant amount: 10-49 group, $s = 1.74$; 50-92 group, $s = 1.94$.

Because the rate of flow data are not normally distributed, no attempt was made to fit normal distribution curves. Here again, however, the distributions probably do not deviate from normality so much as to invalidate the use of the t- and F- tests.

DISCUSSION

In general, the average intraocular pressures, facilities of outflow, and flow rates of

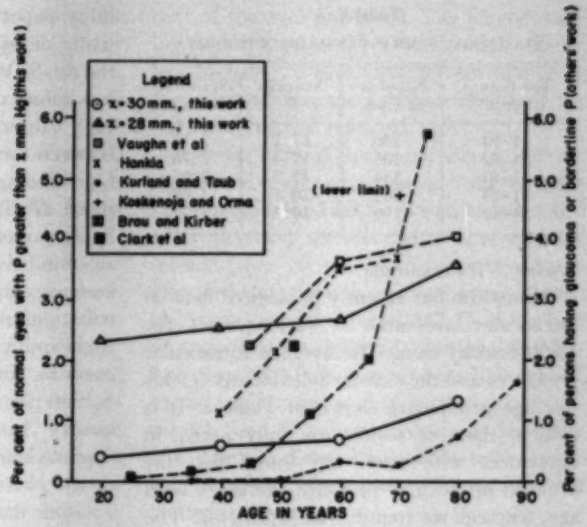

Fig. 5 (Spurgeon, et al.). (a) Percent of normal eyes with pressure greater than 28 mm. and 30 mm. Hg (solid lines). (b) Percent of persons having glaucoma or a borderline increase in pressure (broken lines).

our subjects do not change significantly with increasing age. The distributions widen, however, for all three tonographic values as age increases. In other words, more of the extreme values are found among older people, most of whom, however, show tonographic values that are perfectly normal.

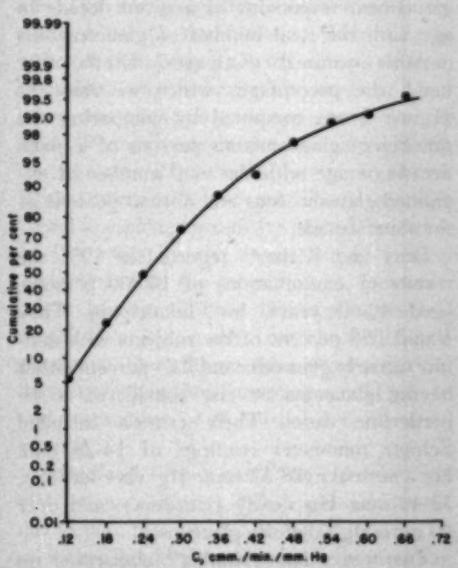

Fig. 6 (Spurgeon, et al.). Test for normality, age group 50 to 69 years.

The changes with age of the intraocular pressure distributions are particularly interesting. As shown by the solid line graphs of Figure 5, the percentage of eyes having pressures over 28 mm. increases with age. The actual percentage in the total population, of eyes having pressures greater than 28 mm. or 30 mm. Hg is probably somewhat higher than shown by the curves. There are two reasons for this: (1) no subjects having intraocular pressure greater than 30 mm. were considered in our study, and (2) it is possible that some of the subjects whose pressure was normal at the time of measurement had abnormally high pressures at other times. If our sample is taken as representative of the population of this area, then the percentage of eyes with pressure greater than 28 mm. Hg ranges from at least 2.4 percent at age 20 years to at least 3.6 percent at age 80 years.

In the literature one occasionally sees statements to the effect that tonometry should be routine on all patients older than 40 years. The solid curves of Figure 5 do not indicate any sudden change in incidence of high pressure at this age, but indicate, rather, that tonometry should be a routine part of the examination of any eyes, regardless of age. This has been pointed out repeatedly by

TABLE 3

STATISTICAL SUMMARY OF ALIMUDDIN'S DATA

Age Group (yr.)	Number of Eyes	Average P	Standard Deviation
11–20	230	17.1	2.15
21–30	206	17.3	2.76
31–40	164	19.0	2.97
41–50	201	20.1	3.08
51–60	199	20.7	3.27

Ascher,[6] for example.

Alimuddin has recently published[5] data on intraocular pressures at various ages for 1,000 healthy eyes. The average intraocular pressures and the standard deviations for his five age groups are shown in Table 3. It is evident that his results are only partly in agreement with ours. He found that the average intraocular pressure increases with age, whereas we found[1] that it does not. The standard deviations for his data increase with age, in agreement with our results. However, plots of his cumulative frequencies on probability paper show that his data are not normally distributed, for any age group or for the totals. With increasing age he finds fewer low values of intraocular pressure, and more high values. The reasons for the differences between the two sets of data are not known. Our findings, however, are in accord with those of Glees,[7] who studied 5,392 eyes of 2,696 persons of all ages. He found no correlation between normal intraocular pressure and age, and considered the frequency distributions of eye pressures to be binominal. Suda and Kiritoshi[8] measured the intraocular pressure in 3,174 normal eys, using the Schiøtz tonometer. They stated that the frequency distributions of pressures are only "approximately binomial," and found no correlation between the tension and age.

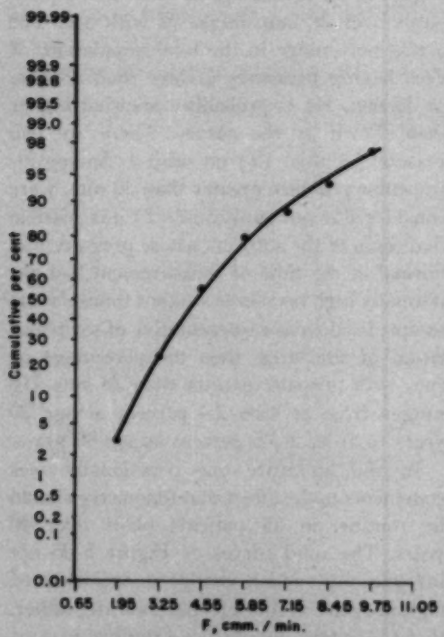

Fig. 7 (Spurgeon, et al.). Test for normality, age group 50-69 years.

Other data from the literature which can be considered in connection with our findings are the results of the various glaucoma surveys that have been made previously. Duke-Elder[9] and Sugar[10] have presented summaries of data from the literature on incidence of glaucoma. The percentages that they give are computed by comparing the number of glaucomatous persons of a given decade in age with the total number of glaucomatous persons examined (of all ages). On the other hand, the percentages which we show in Figure 5 are computed by comparing the number of glaucomatous persons of a given decade of age with the total number of examined glaucomatous and normal persons of the same decade.

Brav and Kirber[11] reported in 1951 the results of examinations of 10,000 persons, aged 40-65 years, in Philadelphia. They found 1.53 percent of the subjects with definite or early glaucoma and 2.24 percent either having glaucoma or else considered to be borderline cases. Their criteria included Schiøtz tonometer readings of 14-28 mm. Hg (normal), 28-32 mm. Hg (borderline), 32-40 mm. Hg (early glaucoma), and over 40 mm. Hg (definite glaucoma).

Two years later Hankla[12] elaborated on these data, most of the subjects being in the

age range 35-65 years. From her data, the percentage of persons having glaucoma or borderline intraocular pressure (28-30 mm. Hg, Schiøtz) are plotted for comparison as one of the dashed curves in Figure 5.

Buesseler, Andrews, Schreuder[13] described the glaucoma detection program at Wright-Patterson Air Force Base in 1952. Seven cases (2.9 percent) of abnormally high intraocular pressure were found in 238 persons over 40 years of age, using the Berens-Tolman ocular hypertension indicator.

Clark, Bancroft, Allen, and Wang[14] studied the incidence, in 1952-1954, of visual defects in two rural counties of Mississippi and a rural parish of Louisiana. In one county the examination included Schiøtz tonometry on adults over 40 years of age. In the other areas such measurements were made only when indicated. Pooling the data of the three areas, they found a glaucoma rate of 0.26 percent. All types of glaucoma are included in their data. Of 30,571 persons examined, 38 had chronic simple glaucoma, eight others had absolute glaucoma, and in three cases the type of glaucoma was doubtful. The low over-all glaucoma rate is accounted for by the fact that their samples had a higher proportion of school children than there was in the total population. Glaucoma was found in each age group over 10 years. For persons in the age range 60-69, the rate was 2.0 percent. For persons over 70 that rate was 5.7 percent. The percentages found in each age group are plotted in Figure 5. The rates by age group are in general agreement with the other figures. However, it is to be expected that the sampling method would give low results for chronic simple glaucoma. The authors themselves point out that any bias in their sampling would lead to underestimation of the visual defect rate in general.

Zeller and Christensen[15] reported in 1954 that of more than 1,000 determinations of intraocular pressure made by 12 interns, 60 eyes were found to have pressure higher than 25 mm. Hg (Schiøtz). This is about six percent of the eyes examined. The percent having pressures over 28-30 mm. would doubtless be lower. These data are not plotted in Figure 5 because the age distribution of the subjects was not reported.

Late in 1954 Wolpaw and Sherman[16] described the results of a glaucoma survey made in Cleveland. Of 12,803 persons tested, 1,635 (12.8 percent) were found to have elevated tension (over 30 mm. Hg Schiøtz, or 40 mm. Hg McLean) in one or both eyes. Proven glaucoma cases numbered 240 (1.87 percent of those tested), with 23 additional borderline cases. No follow-up was possible on 307 of the 1,635 cases, so that, as the authors observed, the 1.87 percent figure is undoubtedly low. The age distribution of the total sample was not given; consequently the data are not plotted in Figure 5.

Vaughan, Asbury, Hoyt, Bock, and Swain[17] published in 1955 the results of a glaucoma survey of 1,000 hospital patients, aged 40-89 years. Their results are included in Figure 5. Between the ages of 40 and 60 years they found that the incidence of glaucoma (counting glaucoma suspects) was about two percent. Above 60 years, however, the incidence rose well above the two percent figure that is often quoted.

In the discussion of the paper by Vaughan, et al., Ellis reported the results of a glaucoma study in a Los Angeles suburb, in which 21 new, definite glaucomas and nine suspected glaucomas were found in 700 people examined. This is 3.0 percent, or 4.3 percent if the suspected cases are included. The results are not shown in Figure 5 because no ages were given.

Koskenoja and Orma[18] (1955) determined the ocular tension (Schiøtz) in 600 persons over 64 years of age, thought to be typical of geriatric patients who visit general practitioners in Finland. They used the same diagnostic criteria as Brav and Kirber, except for the provocative test. Ninety-six persons (16 percent) were found to have pressures greater than 25 mm. Hg in one or both eyes. Four of these were cases of secondary glau-

coma and 37 cases were later found to be normal. Fifteen cases (2.5 percent) of definite, previously undiagnosed primary glaucoma were established, plus 13 cases of previously diagnosed glaucoma (total 4.7 percent). This is plotted as a single point at age 70 years in Figure 5. It is a lower limit, since not all of the 96 persons were examined further. If the borderline cases are included, a possible 52 (8.7 percent) had glaucoma.

The latest studies of this type were reported in 1957. Kurland and Taub[19] studied the frequency of glaucoma in Rochester, Minnesota. Their percentages, plotted in Figure 5, are lower than those reported by others. This is explainable by the fact that only cases of diagnosed glaucoma were included, the total number of glaucoma cases in the community remaining unestimated.

Gradle and Downing[20] described a series of glaucoma case-finding experiments in California. In Alhambra, 2.6 percent of the persons tested were found to have glaucoma. In Glendale, 3.3 percent had glaucoma. A later case-finding project in the same city gave a figure of 2.1 percent. A similar project in Santa Barbara showed seven percent of the number tested to have glaucoma. The criteria used were visual acuity, fundus examination, tonometric testing, and field determination. All of the cases reported were newly diagnosed, that is, the subjects were not previously aware of their disease. The relation between age and tonometric results was not reported, except that in Santa Barbara the median age was high—63 years. The high incidence (seven percent) in this community of older people confirms the other results in Figure 5, namely that the frequency of glaucoma increases with age.

Smillie, Roth, Blum, and Gates[21] determined the intraocular pressures of 1,054 patients over 40 years of age, admitted nearly consecutively to a general hospital in Michigan. They found 10 known cases of glaucoma and 55 new suspects having pressures (Schiøtz) of 26 mm. Hg or more on the 1948 scale. The two groups together constitute 6.2 percent of the total. Further studies were made on 47 of the suspects. Glaucoma was established in a total of 18 cases, or 1.7 percent.

For several reasons, the curves in Figure 5 give only order of magnitude. As is well known, the Schiøtz tonometer values are not the same as manometric pressure values. The tables for use with the Schiøtz tonometer have been changed throughout the years, and these changes make comparisons difficult when the work of different investigators is compared.

There are other reasons, too, why the curves give only order of magnitude. In most of the cases cited the orginal age data, if given at all, are grouped rather than given in detail. Although points are plotted at definite ages, therefore, their locations should be regarded as approximate.

Moreover, it can be argued that in the samples studied, the percentage of glaucomatous eyes found should be regarded as minimum figures. The actual proportions of eyes with abnormally high intraocular pressure are probably greater than found, for it is well known that glaucomatous eyes can have pressures well within normal limits at some hours of the day. If they were measured at a time when the pressures were normal, and the glaucoma was not found by field examinations or some other diagnostic test, then these eyes would not be counted as glaucomatous. Early glaucoma and so-called low-tension glaucoma can also be missed.

It can be questioned whether any of the samples studied were really completely representative of the actual population. It seems to be extraordinarily difficult, in studies of the rate of occurrence of glaucoma, to devise a practical sampling procedure that is free of bias. In our study, for example, the subjects were well distributed by age, race, and sex, but intraocular pressure values above 30 mm. Hg were excluded in order that only normal eyes would be in the sample. In samples drawn from industrial groups, most

persons over 65 years of age are excluded and for reasons of economy persons under 35 may be discouraged from taking the tests. Persons not working on account of defective eyesight or other disability, or on account of sex, are also not included in such studies. In surveys such as the one in Cleveland there may be a preponderance of females because the men are at work. Many older people who have difficulty in getting around would also be excluded in such work. In studies based on medical records of a community it is questionable whether all persons, or even a representative sample have their intraocular pressures measured. In all of the studies discussed, therefore, the sampling bias is such that the estimated incidence of glaucoma is probably too low.

A statistically unbiased study made on a representative sample of the population would have to be based on census figures. Each age, race, and sex should be represented in the sample in about the same proportions as in the general population. All pressures found should be included. Pressures should be determined more than once, preferably at those times of day when they are likely to be highest. Borderline cases should perhaps be checked with more than one tonometer. The study of a large sample selected in this way would be long, difficult, and expensive. No one has yet suggested a practical way of accomplishing it.

In view of the difficulties encountered, the agreement in the results of the workers cited is quite good. It seems likely that the often quoted figure of two percent of persons over 40 years of age having glaucoma is conservative. For persons aged 40 it may be about right, but at age 60 the figure is probably 3.6 percent or more; at 80 years it may be 4.0 percent or higher.

Our data, and rough extrapolation of the data of Vaughan and of Hankla suggest that the percentage of persons having high intraocular pressure or glaucoma at ages below 40 years is by no means negligible. This suggests that tonometry should be done routinely as part of the eye examination even on younger patients. Ascher has pointed this out repeatedly, for example, in his discussion of the paper by Brav and Kirber.[11] Our findings thus confirm his statements.

The normal curves fitted to our data taken from normal subjects lead to the prediction that the percentage of persons having intraocular pressures greater than 28-30 mm. Hg increases as age increases. The prediction is confirmed by the figures of Hankla, of Vaughan and co-workers, of Kurland and Taub, of Clark, of Koskenoja and Orma, and of Gradle and Downing, all showing that the percentage of persons having glaucoma or borderline pressure increases as age increases. These facts can be explained if we consider the anatomic changes in the chamber angle and its vicinity which occur with increasing age. For example, the work of Dvorak-Theobald and Kirk[22] and of Teng, Katzin, and Chi[23] on serial sections of glaucomatous eyes and Rédmond Smith's work on neoprene casts of glaucomatous eyes[24] all indicate impairment of the aqueous drainage channels.

An additional factor which could affect the relations between intraocular pressure and age in such a way as to increase the percentage of persons with high apparent intraocular pressure is the change of scleral rigidity with age. Friedenwald discussed this problem[25] in 1937. He stated that, beginning in the fifth decade of life, "an increasing rigidity of the ocular coats is characteristic of advancing age." Examination of his frequency curves for scleral rigidity at various age levels, however, reveals an additional fact of great interest. The frequency curves for the age groups 15-30 and 31-50 years were about the same in appearance, and the distributions appeared to be normal. The frequency curve for the age group 51-60 years consisted of a large, normal-looking part having about the same mean and spread as the younger age group, plus a small, humplike extension of the tail of the curve on the high side. In other words, the distribution curve for the

group 51-60 years is bimodal: there are actually two distributions, one normal (in both senses of the word) and one that is possibly normal in the mathematical sense but abnormally high in the biologic sense. For the age group over 60 the second, abnormally high distribution is even more pronounced. It would be a most interesting study to determine whether there is any connection between the high scleral rigidity in the "abnormal" group of the older eyes, and the onset of glaucoma.

Since the normal curves for the distribution of intraocular pressures are symmetrical, it follows that the percentage of persons having abnormally low pressures (less than 13-14 mm. Hg) also increases with age (fig. 4). This is a new and unexpected physiologic fact in geriatrics, worth further study since the reason is unknown.

Magitot stated[26] that intraocular pressure in children's eyes is higher than in eyes of aged persons and suggests that low ocular tension in aged individuals can be considered a sign of poor peripheral circulation (in the choroid). Increased scleral rigidity, however, may mask this effect. Other possible causes for low intraocular pressure also come to mind: wider or shorter outlet channels from Schlemm's canal (not probable); operation of Bárány's mechanism in the trabecular spaces; low rate of production of aqueous; decrease of viscosity of the aqueous humor; decrease of systemic or episcleral venous pressure; and decreased blood volume in the uvea.

Summary

The spreads of tonographic values for normal eyes increase in general with increasing age, that is, in the older age groups there are more persons whose tonographic values differ widely from the averages. For intraocular pressures the wide deviations may be on both the high and the low sides.

The increase with age of the percentage of normal eyes having intraocular pressure higher than 30 mm. Hg is of interest when compared with the results of various glaucoma surveys. The incidence of glaucoma found in all of these surveys is probably too low. High intraocular pressures are found at all ages. Accordingly, tonometry should be a routine part of the eye examination at all ages.

The increase with age of the percentage of eyes having intraocular pressures less than 13 to 14 mm. Hg is a new finding in geriatrics. Several possible explanations can be given, but the real reason is still unknown.

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References

1. Boles-Carenini, B., and Cambiaggi, A.: Are aqueous humor dynamics influenced by aging? Am. J. Ophth., **44**:395-401, 1957.
2. Weinstein, P.: Glaucoma: Pathology and Therapy, St. Louis, Mosby, 1953, p. 287.
3. Boles-Carenini, B.: Principi, Tecnica e utilita della tonografia, Boll. ocul., **34**:403-432, 1955.
4. Kenney, J. F.: Mathematics of Statistics. New York, Van Nostrand, 1939.
5. Alimuddin, M.: Normal intraocular pressure. Brit. J. Ophth., **40**:366-372, 1956.
6. Ascher, K. W.: Discussion of paper by Brav and Kirber. Tr. Sect. Ophth. A.M.A., 1951, p. 61.
7. Glees, M.: Statistische Berechnungen über die Hoehe des normalen Augeninnendrucks. Arch. f. Ophth., **153**:356-358, 1952.
8. Suda, K. and Kiritoshi, Y.: Distribution of normal intraocular tension. Acta Soc. ophthal. jap., **57**: 886-892, 1953; Abstract: Ophth. Lit., **7**:373, 1953.
9. Duke-Elder, S.: Textbook of Ophthalmology. London, Kimton, 1941, v. 3, pp. 3329-3330.
10. Sugar, H. S.: The Glaucomas. St. Louis, Mosby, 1951, p. 126
11. Brav, S. S., and Kirber, H. P.: Mass screening for glaucoma. J.A.M.A., **147**:1127-1128, 1951.
12. Hankla, E. K.: Glaucoma case finding in Philadelphia. Public Health Rep., **68**:1059-1064, 1953.
13. Buesseler, J. A., Andrews, A. C., and Schreuder, O. N.: Glaucoma detection program at Wright-Patterson Air Force Base. Tr. Am. Acad. Ophth., Nov.-Dec., 1952, pp. 982-984.

14. Clark, W. B., Bancroft, H., Allen, J. H., and Wang, Y. L.: Incidence of visual defects in two rural counties of Mississippi and a rural parish of Louisiana; A study sponsored by the Nat. Soc. Prev. Blindness, et al.
15. Zeller, R. W., and Christensen, L.: Routine tonometry as part of the physical examination. J.A.M.A., **154**:1343-1345, 1954.
16. Wolpaw, B. J., and Sherman, A. W.: The Cleveland glaucoma survey, Sight-Saving Rev., **24**:139-144, 1954.
17. Vaughan, D. C., Asbury, T., Hoyt, W. F., Bock, R. H., and Swain, J. M.: Glaucoma survey of 1,000 hospital patients, Tr. Pac. Coast Oto-Ophth. Soc., 1955, pp. 99-105.
18. Koskenoja, M., and Orma, E.: Routine tonometry for glaucoma detection in geriatric patients. Geriatrics, **10**:362-365, 1955.
19. Kurland, L. T., and Taub, R. G.: The frequency of glaucoma in a small urban community. Am. J. Ophth., **43**:539-544, 1957.
20. Gradle, H. S., and Downing, B.: Community-wide glaucoma case finding, Sight-Saving Rev., **27**: 78-82, 1957.
21. Smillie, J. W., Roth, F. D., Blum, F., and Gates, L. K.: Glaucoma detection in a general hospital. Am. J. Ophth., **44**:20-23, 1957.
22. Theobald, G. D., and Kirk, H. Q.: Aqueous pathways in some cases of glaucoma. Am. J. Ophth., **41**:11-21, 1956.
23. Teng, C., Katzin, H. M., and Chi, H. H.: Primary degeneration in the vicinity of the chamber angle: Part II. As an etiologic factor in wide-angle glaucoma, Am. J. Ophth., **43**:193-203, 1957.
24. Smith, R.: Blood in the canal of Schlemm. Brit. J. Ophth., **40**:358-365, 1956.
25. Friedenwald, J.: Contribution to the theory and practice of tonometry, Am. J. Ophth., **20**:985-1024, 1937.
26. Magitot, A.: Physiologie Oculaire Clinique. Paris, Masson, 1946, p. 114.

SPONTANEOUS INTERNAL SCLERAL RUPTURES

AND THE SPLITTING OF THE CORNEA-SCLERA

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As a rule ruptures of the sclera are caused by injuries. Sometimes these are direct ruptures; more frequently, however, they are indirect ruptures and are located internally and above, about two mm. behind the limbus and run parallel to and behind it.

The fact that the indirect ruptures are located in the nasal and upper quadrant of the globe is explained as follows: the region of the internal scleral sulcus is an especially weak part of the sclera, weakened the more because here the anterior ciliary vessels run from Schlemm's canal backward and outward. Indirect ruptures follow this course of the vessels, and are nearly always found internally and above.

This may be explained by the fact that the trochlea and the spina trochlearis, around which the tendon of the superior oblique muscle runs, are situated here; these form a hard protuberance at the inner upper orbital wall, next to the incisura supraorbitalis. If an eyeball—for instance, by a blow with a fist—is pressed into the orbit, the cornea is flattened and the weak region behind the limbus is pressed against the trochlea and is indented by it. The considerably increased pressure of the eye causes the eye to burst in this indented region. Bell's phenomenon plays a special rôle in these cases because the region of Schlemm's canal and the trochlea are approached by the upward movement of the globe.

Beside these traumatic ruptures spontaneous ruptures of the sclera may also occur. They are very rare; I found only a few cases in the literature.

Sometimes the sclera is lacerated far behind. Mathewson[1] (1932) gave a very short description of four cases: (1) A 13-year-old boy after operation for cataracta perinuclearis; (2) a 60-year-old woman, who was operated by Elliot trephining for a subacute glaucoma; (3) a 40-year-old woman with a

spontaneous scleral rupture; of these cases nothing was noted of the localization of the rupture. Only in the fourth case, a 50-year-old woman, who had severe pains for six years, a histologic examination was made and it was stated that the sclera was ruptured all around its equatorial region.

Another very unusual case was published by Michel[2] (1948). A 10-year-old girl had high myopia (20D.); without any injury she suffered severe pain for several days. Then ptosis, considerable enophthalmos, and immobility of the globe set in. The iris had fallen back considerably and the retina was detached below and behind. Tension was 0. After three months the lids and the position of the globe and immobility were normal again, the tension nearly equal to the other eye; the retinal detachment was still present. Vision was hand movements at 50 cm. Michel assumed a spontaneous rupture of the sclera but he did not discuss the localization of it.

Spontaneous scleral ruptures are somewhat more frequent in the region of the sulcus sclerae. Ernst Fuchs[3] (1912) examined a 62-year-old woman with such an ectasia anatomically; he also examined an eight-year-old girl who had an ectasia of the nasal half of the cornea due to a rupture of the corneal-scleral region two months after being hit by a stone.

Stölting[4] (1913) described a 14-year-old girl whose right eye was bigger than the left eight days after birth. Later on a staphyloma developed all around the cornea and below a leukoma of the cornea was found. The anatomic examination revealed a rupture in the internal scleral sulcus from which a slit extended posteriorly between the inner and outer layer of the scleral lamellae.

Richman[5] (1936) noted a 25-year-old Negress who had a binocular chronic iritis with tuberculous nodules. Spontaneously the sclera ruptured at the limbus and the iris prolapsed there between the 2- and 5-o'clock positions. The vision became better (20/70) because the very narrow pupil had become wider due to the prolapse.

Dejean et Boudet[6] (1951) presented a 64-year-old woman who had severe glaucoma for 14 years. Finally she had very severe pains for 16 days and bloody liquid oozed from the eye for several hours. The eye was ruptured at the corneal-scleral junction. A second patient, a 60-year-old woman, had glaucoma in both eyes due to iris en tomate. As she had poor light sensation in the left eye, she was operated only on the right eye. Fifteen years later she suffered terrific pains in the left eye; blood oozed out and the pain stopped. Here again a rupture was found behind the limbus, below; iris and lens were prolapsed. At the same session similar cases were reported: Jaquet mentioned a rupture of the sclera two days after retrobulbar injection; Carlotti and also Baille reported cases of spontaneous rupture behind the limbus.

Spontaneous internal ruptures of the sclera in the region of the internal scleral sulcus and Schlemm's canal were found several times during my histologic studies. As there are interesting details of the structure and the tectonic of the cornea-sclera I will describe a few of these cases:

Case reports

Case 1

The eye was enucleated on account of retinoblastoma. The anterior chamber was extremely shallow, the iris was lying against the cornea on one side, while on the other side only the peripheral half was adherent. The pupil was maximally wide. The anterior chamber contained a few cells of glioma and phagocytes with pigment; a larger amount of these cells were present in the circumlental space, here more mixed with pigmented phagocytes. The lens was cataractous and calcified in the posterior equatorial parts. Nearly the whole globe behind the lens was filled with partly necrotic tumor masses which had pushed the vitreous forward against circumlental space. The choroid was partly attacked posteriorly by the retinoblastoma; on one spot tumor cells are present on the outside of the sclera in the neighborhood of a small ciliary nerve.

On one side in the region of the internal scleral sulcus the tumor had, by increasing tension, caused a dehiscence of the inner and outer scleral lamellae and a small intercallary staphyloma. Here a pocket was formed by the external scleral lamellae and interiorly by a detached scleral corneal ridge.

The posterior ridge corresponded to the scleral spur. The anterior ridge represented the periphery of the posterior corneal layers. This pocket occupied five mm. of the corneal circumference and had a communication with the interior corresponding with the former internal scleral sulcus.

Into this pocket parts of the ciliary body and ciliary process, dispersed with glioma cells, were pressed (fig 1). The posterior part of this niche had an extension of three mm., the scleral ridge 0.6 mm., the corneal ridge projected 1.2 mm. This pocket or niche was pointed posteriorly and anteriorly. Posteriorly the tear or dehiscence ran along the anterior ciliary vessels. However, the vessel (K) did not seem to be an aqueous vein because it had quite a thick wall, which hardly could be attributed to aqueous veins. Anteriorly the dehiscence extended much further, about twice as much as behind. Two vessels extended from the anterior end of the pocket into the corneal parenchyma.

One can easily recognize here that in the region of the internal scleral sulcus the connection between the inner and outer layers as well as of the sclera and of the cornea was weak and that it did not withstand the increased tension. This, however, is an exception because, in general, the scleral lamellae which form the bottom of the internal scleral sulcus simply yield and no dehiscences occur along the meridional layers of the fibrous capsule of the globe.

A smaller niche but of similar configuration I found in another case of retinablastoma.

CASE 2

The glioma had filled the whole globe and had proceeded into the optic nerve and the intervaginal space; both were considerably enlarged. The uvea was nearly entirely replaced by tumor and was recognizable only by some remains of pigment epithelium and Bruch's membrane. On the temporal side the internal scleral sulcus was not attacked

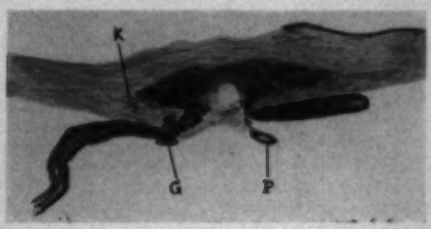

Fig. 1 (Fuchs). Internal scleral rupture. (K) Anterior ciliary vessel. (G) Nodule of glioma retinae. (P) Ciliary process.

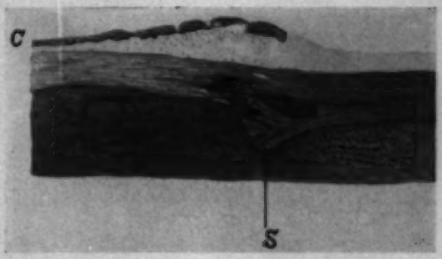

Fig. 2 (Fuchs). Internal scleral rupture and deviation of scleral spur. (S) Scleral spur.

by the tumor. On the nasal side, however, masses of the tumor had invaded the sulcus and were proceeding anteriorly and posteriorly, having separated the outer scleral layers from the inner ones. Here again there was a pocket or niche, of 1.2-mm. length (fig. 2). The scleral spur (S) was pushed inward. Fine slits parallel to the surface ran from the posterior end of the pocket backward in the middle layers of the sclera. In an analogous way strands of fine pigment, tumor cells, and vessels extended from the anterior recess of the pocket one mm. further into the deep layers of the cornea.

In other series of sections of the globe I found considerably wider bulging of the sclera and a much more marked deviation of the scleral spur with the internal scleral lamellae toward the inside of the globe.

Also in this case a pocket is formed in the internal scleral sulcus and it is remarkable how the scleral spur is bent inward. One would suggest that this deviation is caused by the traction of the scleral muscle but the muscle was entirely replaced by the tumor. Thus another cause apparently brought about the deviation, probably the presence of elastic fibers in this region of the internal scleral sulcus. Of this I will speak later.

A similar change as in Case 2, I mean a kind of chipping of the internal scleral lamellae together with the scleral spur, was found in another eyeball with retinoblastoma.

CASE 3

A glioma had filled the whole globe and transformed the content into a tumor. The eyeball was opened during the enucleation at the insertion of a rectus, that is, three mm. of sclera were excised. The opening was filled with blood and tumor. The cornea showed an ulcus (U) with a flat loss of substance and progressive borders. In front of Descemet's membrane, on both sides of the region of the ulcus, the cornea was infiltrated by glioma cells (G).

The retinoblastoma had invaded the internal

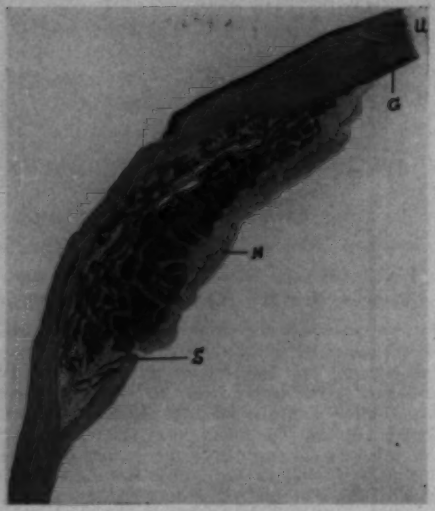

Fig. 3 (Fuchs). Internal scleral rupture. (S) Scleral spur. (G) Glioma cells. (N) Necrotic tumor masses. (U) Progressive border of ulcus serpens.

scleral sulcus and had pushed the thin roof of internal scleral sulcus together with the neighboring cornea outward. More posteriorly the inner scleral lamellae (fig. 3) had become detached from the outer ones and a slit of 2.5-mm. length had been formed. This slit was filled anteriorly by tumor, posteriorly by isolated tumor cells, edema, and some lacerated bundles of connective tissue.

In the two former cases a pocket with an anterior and a posterior slit was present. In this case an intercallery staphyloma was present and the scleral spur (S) with a considerable part of the internal lamellae was separated and had deviated interiorly to a considerable extent.

In the part of my collection which I have in New York I have 45 cases of retinoblastoma and in three of these cases such internal lacerations were present. In two other cases I found an insignificant chipping of the scleral spur and the inner lamellae; the dehiscence in the posterior part of the intercallary staphyloma amounted only to 0.2 mm. and from this small laceration there was no continuation backward. In five cases the retinoblastoma had perforated the sclera near the cornea without a similar tear.

I found a similar dehiscence of the external layers of the sclera from the internal layers of the cornea in eyes containing other tumors.

CASE 4

This case shows a diktyoma; it has been published by Böck[3] (1929).

The ciliary body of a six-year-old child was covered on one side by a thin layer of tumor cells which extended to the equator of the lens (fig. 4). The internal scleral sulcus was considerably enlarged (three mm.) and external scleral lamellae bulge to the extent of 1.5 mm. Here the internal lamellae of the sclera were distended two mm. from the external ones and the same was true for the posterior lamellae of the cornea (0.8 mm). The scleral spur (S) together with the inner lamellae was deviated inward, apparently due to the traction of the ciliary muscle. The groove of the internal scleral sulcus, enlarged by the two recesses anteriorly and posteriorly, contained a few small flaps of tumor which were separated by empty spaces. The anterior (T) and posterior recesses were filled by loosened particles of tumor and isolated bundles of corneal and scleral fibers. The tumor showed the typical structure of a diktyoma: some long garlands of cells which looked exactly like the unpigmented epithelium of the ciliary body, some nerve tissue with considerable malformation, abundance of nuclei, neuroglia, and rosettes. At the anterior edge of the intercallary staphyloma the

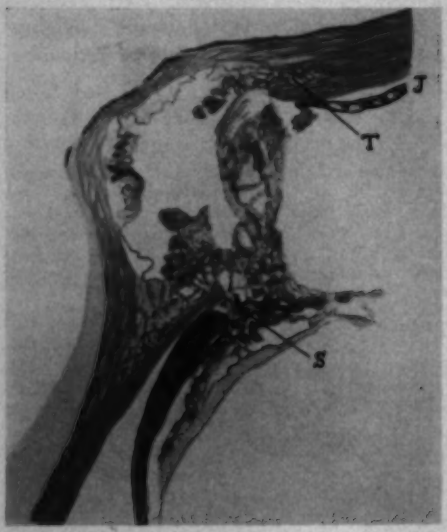

Fig. 4 (Fuchs). Internal scleral rupture. (S) Scleral spur. (T) Tumor cells (diktyoma). (I) Iris.

iris (I) became visible and ran against the interior of the eye; it was very thin, atrophic, and covered by endothelium.

In contrast to the recess in the area of the intercallary staphyloma in cases of glioma we find the space here only partly filled by tumor masses. It was not the diktyoma which had caused a liquefaction of the tissue and thus ectasia and splitting of the sclera (cornea) but here the glaucoma had lacerated the apparently poor connection between the inner and outer lamellae. This laceration and the formations of niches were not present all around, as in the other cases; only at one point of the ectasia. Seemingly the connection of the different layers of the lamellae of the corneal sclera is not always the same.

The iris was entirely separated from the ciliairy body; the inside of the intercallary staphyloma was not covered by pigment, as one may observe in other staphylomas. The staphyloma was not formed gradually and the root of the iris was not stretched and was not atrophic and reduced to a pigmented layer. The tissue here was apparently really lacerated and an iridodialysis had taken place.

Case 5

This case shows a hemangioma of the choroid; it was published by Bergmeister[8] (1911).

A 26-year-old woman suffered an inflammation of the left eye four years ago and became blind. The eye was enucleated because of pain.

The iris was pressed against the cornea and contained a few very large blood vessels. The anterior chamber was abolished. A large intercallary staphyloma was formed by a considerable widening of the internal scleral sulcus. Only a step corresponding to the scleral spur was found; however, there was a considerable splitting of the sclera (fig. 5) and the scleral spur (S) was deviated, as were the strong scleral lamellae, inward and formed a ledge one-mm. wide which outlined a niche or pocket posteriorly. The scleral lamellae forming the staphyloma anteriorly were quite thin. Ciliary process (Z) and a part of the muscle were pressed into the pocket between the split parts of the sclera. Without doubt the increased pressure had pushed the ciliary body around the scleral spur into the recess. On other slides the corneal lamellae were split, as well, and one found a condition similar to that shown in Figure 2, as a small entrance led into a recess which was filled by a ciliary process. The lens was pressed against the iris and the cornea; the retina was pressed against these two in such a way that the retina even extended into the pocket. A large angioma of the choroid had led to subretinal glaucoma.

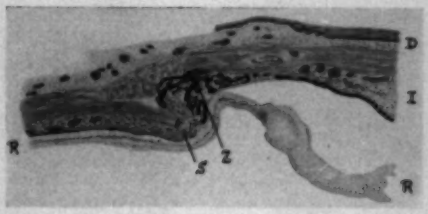

Fig. 5 (Fuchs). Internal scleral rupture. (R) Retina. (S) Scleral spur. (I) Iris with enlarged vessels. (Z) Ciliary process. (D) Degenerative pannus.

The sclera in the neighborhood of the internal scleral sulcus was slit and the scleral spur forming a ledge had deviated inward, as in Case 4. Probably the deviation of the scleral spur in this case was not due to a traction of the ciliary body as a part of it lay in the recess. It seems more likely that here, as in Case 4, the elastic system of the scleral spur had contracted and caused this deviation. The enormous pressure in the subretinal space by which the lens and the retina were pressed against the anterior wall of the globe was remarkable.

All the cases enumerated concern tumors with increase of tension and a seemingly high degree of yielding of the sclera. I have seen similar lacerations of the sclera in the region of the internal scleral sulcus in which no tumor, only glaucoma, was present.

Case 6

A man, aged 40 years, suffered a corneal injury with scissors when he was three years old. The eye became blind and enlarged (buphthalmos) to 30 mm. in length. There was a ciliary staphyloma above the cornea. The scar of the lower cornea was connected by a membrane with the cataracta membranosa. The anterior chamber was of medium depth. The iris was partly adherent to the periphery of the cornea and quite atrophic.

The internal scleral sulcus was not changed below while above the internal scleral sulcus was very enlarged; only the exterior scleral lamellae were left (fig. 6). A considerable recess (2.5 mm.) was formed and a slit (one mm.) extended into the cornea. Here one did not find a smooth interior surface of the intercallary staphyloma, as elsewhere, for connective tissue fibers looking like

Fig. 6 (Fuchs). Internal scleral rupture in buphthalmos. (S) Scleral spur.

shreds, were present in the interior coming from the sclera and cornea into the cavity. The region of the scleral spur (S) was somewhat deviated. In some sections a real cavity at the end of the slit was found in the cornea, different from the cases with tumor where narrow pointed slits were present. Posteriorly not much of the ciliary staphyloma could be seen in this region of internal laceration of the sclera. In the places where the region of the internal scleral sulcus was normal, again the ciliary staphyloma was much more marked and the sclera in its entirety thinner and more projected.

There was a different kind of laceration of the corneal sclera here, as there was no smooth excavation at the margin of the cornea and pieces of the original connective tissue could still be seen. This resembled threadbare cloth. This was especially interesting because it is generally agreed that an intercallary staphyloma is due to secondary glaucoma which has just bulged and attenuated the region of Schlemm's canal. It is evident, however, in this case, that not only simple ectasia of the tissue is present but that also a number of bundles have been lacerated. Here is found a slight inward deviation of the scleral spur (S).

The largest extension of a splitting of the cornea-sclera I saw in a case sent to me by Dr. Bertha Klien, for which I express my sincere thanks.

Case 7

A 12-year-old child had hurt its eye with glass and had become blind from glaucoma. The corneal center showed a barely visible old scar which was recognized by some scar tissue elevated over the corneal surface and by some folds of Descemet's membrane posteriorly. The cornea was quite thick but on both sides, there was a large intercallary staphyloma of about five mm. (fig. 7). The external scleral lamellae were quite attenuated; the sclera and cornea were torn in such a way that, interiorly, considerably elevated ledges were formed. On one side the ledge projected anteriorly and posteriorly 1.5 mm.; on the other side there was no anterior ledge. No pocket had formed. However, posteriorly there was a ledge. Both posterior ledges deviated inward considerably, especially on the right side. The ciliary body was attached at the edge of the ledge, which was originally the scleral spur.

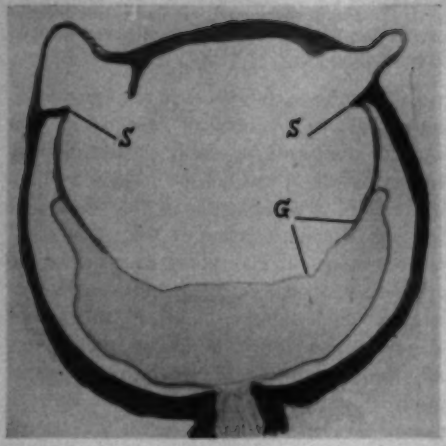

Fig. 7 (Fuchs). Internal scleral rupture. (S) Scleral spur. (G) Anterior limiting layer of vitreour body covered by epithelium and pressed backward.

The posterior surface of the cornea, the staphyloma, and the ciliary body were covered by a thin epithelial layer. This epithelial layer proceeded from the ciliary body to the anterior limiting layer of the vitreous body. The vitreous body, also detached posteriorly, was pushed backward and transformed to a thin membrane (G). The retina lying upon the choroid was connected with it. The papilla was quite atrophic but without glaucomatous excavation. The ciliary body, together with the choroid, was detached artificially; the lens and ciliary process were absent.

The case is quite interesting because of the absence of the iris and the lens which were probably expelled during the injury (probably a large incised wound). Thus an unusual epithelial inngrowth was possible as the anterior and posterior chamber became a cavity of 12-mm. depth, extending from the cornea onto the vitreous. Still more interesting is the broad splitting of the sclera and the cornea which extends nearly all around. The inward deviation of the interior ledge is particularly striking here and becomes especially marked by the straining of the ciliary

body which is artificially detached.

All the cases here described had an ectasia due to glaucoma; the first ones due to tumor, the latter ones due to injury. However, such an internal scleral rupture can also develop occasionally without prolonged secondary glaucoma, as is demonstrated by the next case.

CASE 8

A boy, 14 days of age, died of sepsis after infection of the umbilical cord. An abscess of the kidney and a metastatic ophthalmia with ring abscess of the cornea had occurred.

The cornea had lost the epithelium and was pervaded by pus cells. On both sides in front of Descemet's membrane leukocytes had accumulated at a considerable distance from the limbus. The anterior chamber was filled with exudate containing necrotic pus, fibrin, and cocci. On one side, the region of the Schlemm's canal was liquefied and dissolved so that an intercallary staphyloma developed. The thinned and bulging scleral lamellae outlined a hole, a cavity three mm. in length, which was connected with the anterior chamber by an opening of two mm. The scleral part of the cavity was full of fluid, the internal lamellae were deviated inward, and formed a niche with the external lamellae. The cavity extended much further into the medial part of the cornea; here the internal corneal lamellae projected and some were dissolved into isolated, infiltrated threads of connective tissue. In the cavity there were shreds and pieces of necrotic tissue and exudate (fig. 8).

The ciliary body was almost completely separated from the scleral spur (S); it was connected with it by a few fibers. However, it slipped backward and a slit extended from the anterior chamber between the external bundles of the muscle and the corona ciliaris. Such slits are frequently found as sequels of trauma. In the depth of this slit there were masses of cocci between the muscular bundles. The part of the iris in the vicinity of the abscess of the sclera was entirely necrotic—the tissue without nuclei and filled with colonies of cocci and the pigment epithelium almost all dispersed. The other parts of the iris were filled with pus cells but were not necrotic; apparently the toxic action of the metastic abscess of the sclera had not extended so far. The epithelium of the lens capsule had vanished. The circumlental space was filled with exudate and the anterior surface of the vitreous was covered with a considerable mass of cocci. The ciliary body was completely covered with necrotic exudate.

Fig. 8 (Fuchs). Internal scleral rupture and metastatic abscess. (S) Scleral spur.

In this case of metastatic ophthalmia the scleral abscess at the limbus had led to laceration of the tissue similar to the cases in which prolonged secondary glaucoma was the cause. Here a cavity was formed by liquefaction and it extended anteriorly and posteriorly into the sclera and the cornea. Also in this case, the internal scleral lamellae were deviated inward, the internal corneal lamellae, however, were not, because, perhaps, the deeper marginal corneal lamellae were infiltrated (ring abscess). This probably had diminished or annulled the elastic power of Descemet's membrane. It is clear that the connection between the stroma and Descemet's membrane would be diminished if pus were deposited. Also, in this case, the walls of the cavity were jagged and shreds projected from the cornea against the inside of the cavity.

A synopsis shows that in all these cases the sclera was lacerated in the region of the internal scleral sulcus in such a way that the sclera was not lacerated from the inside to the outside but that the external scleral lamellae were lacerated and deviated from the internal portion. These lacerations, originating in the internal scleral sulcus, were sometimes quite broad at their start but diminished in size anteriorly and posteriorly. One can find these lacerations only on circumscribed spots on the internal scleral sulcus (Case 6). They may also be present in large areas of the circular internal scleral sulcus and were largest in Case 7. At the same time the external lamellae roofing the internal scleral sulcus are considerably thinned and more or less ectatic so that the clinical picture of an intercallary staphyloma is present.

Certainly the internal scleral ruptures are

Fig. 9 (Fuchs). Normal filtration angle, elastic fibers stained. (E) Circular bundles of sclera surrounded by elastic fibers.

mostly due to glaucoma. Considering the cases of retinoblastoma alone, one may perhaps be tempted to believe that dehiscences and splits between the external and internal scleral and corneal lamellae are caused by an active invasion by tumor cells. Other tumors show that this is not the case; in the case of diktyoma there are only a few small strands of tumor cells in the pocket and, in Case 4, the choroidal hemangioma had no direct relation to the place of the scleral rupture. The other cases of secondary glaucoma show that increased tension had for the most part caused the separation of the two layers of connective tissue.

Why does secondary glaucoma lead to this kind of laceration and separation at this place? The following are decisive factors in the anatomic structure of this region:

In the region of the internal scleral sulcus, the external bundles of the sclera run meridionally, that is, the bundles are cut longitudinally in the sagittal section. The internal layers of the sclera represent cross sections of equatorial, circular running bundles (fig. 9) in the region of the sclera behind the scleral spur.*

These bundles line the posterior wall of the internal scleral sulcus. Their cross sections appear oval in some specimens; others have the shape of a lance. More posteriorly they become longer because here the bundles apparently deviate from the equatorial course toward the back. The circular bundles extend backward about 1.5 mm. from the scleral spur, as can be seen upon examination of slides where the elastic fibers are stained. The accumulation of the circular bundles occupies the inner third or quarter of the sclera. The bundles are separated by elastic fibers and so, in specially stained specimens, they become particularly conspicuous. In the middle and external part of the sclera, there are only a few elastic fibers, while in the internal layers the fibers are strongly developed. It is interesting that the elastic fibers surrounding the cross section of the connective tissue bundles are cut across and not lengthwise. The area of intensively developed elastic fibers does not include the whole region of circulary bundles, but only the innermost layers of the sclera, demonstrating that a zone of cross-sectioned equatorial bundles is also visible on the exterior of the abundant elastic fibers (fig. 9).

The fact that the inner layers of the sclera behind the internal scleral sulcus have a structure quite different from the external layers explains why the external layers are relatively easily separated from the inner layers if considerable pressure is exerted through the scleral canal upon the external layers. Apparently in this region there is no marked interlacing.

It is not possible to find a similar cause for splitting in the cornea. Another factor is

* The term scleral spur is derived from the meridional sections. Salzmann proposed for this term the name "Skleralwulst," translated by E. V. L. Brown (1912) as "scleral roll," because one finds a circular ledge behind the internal scleral sulcus and nothing pointed which would suggest a spur, if the eye was cross-sectioned frontally. It would seem best to stick to the old name, scleral spur, as I am not in favor of creating new names. Anyhow I would consider "Leiste" in German and "ledge" better than "wulst" or "roll" because both the latter words mean something round and the scleral spur represents something sharp but not pointed, easily seen on examination with a probe and a loupe of an anatomic preparation of a scleral spur.

probably active. In the sclera, as in the cornea, there is a deviation of the internal ledge in an inward direction. This is most conspicuous in Case 7. The reason for this inward deviation of the scleral layers is, in some cases, the traction of the ciliary muscle which is inserted at the scleral spur and acts through its muscular tonus; this seems to be considerable in some cases. In the cornea there is only one circular bundle, that is, the limiting ring of Schwalbe. Frequently, it exists only in part and not always that. It is situated at the edge of Descemet's membrane. In rare cases it is developed to a high degree (A. Fuchs[9]) so that it may form a considerable obstacle during a cyclodialysis operation. (The spatula gets stuck and one has to withdraw it.) The ring may even become detached and be pushed, together with Descemet's membrane, into the anterior chamber where it becomes visible. Usually, Schwalbe's ring is not a real ring but is composed of small pieces. It may play a rôle in the tectonic strength of the cornea but not an essential one. In the cases reported herein no marked Schwalbe's ring was present.

More important however is the elastic action of Descemet's membrane which becomes evident in long incised wounds of the cornea and in indirect ruptures, causing considerable inward curvature of the cornea. Long incisions for intracapsular extraction of the lens lead to internal gaping of the wound, as was recently shown by B. Samuels.[10] Therefore, Descemet's membrane may cause deviation of the posterior corneal margin and may sometimes pull the internal lamellae inward and produce the ledge.

As already mentioned, a loose connection between the inner and outer layers in the region of internal scleral sulcus was seen in all the cases. Apparently the connection is not the same in all eyes nor in all spots in individual eyes. Otherwise the internal laceration would be seen more often and the lacerations in the eyeballs mentioned herein would not be seen only on circumscribed spots. Seemingly, there is a certain anomaly of the structure of the sclera in this region.

It may be important that almost always the eyes which are seen are those of children or of persons who either underwent enucleation at this age or were children when the glaucoma set in. Probably in youth when the tissue is not matured and not yet tough, the connection of the different layers of the anterior fibrous capsule of the eye is not as developed as in adults. I have not seen a case of an adult with this type of internal scleral rupture due to secondary glaucoma.

In connection with this point I would like to mention interscleral epithelial cysts. They develop at the limbus, separate the sclera and cornea into two layers, and are lined by epithelium. They reach no farther than two mm. into the cornea. The inner wall of the cyst consists, as a rule, of one half to two thirds of the scleral thickness, while the external wall is formed by the remaining portion. Salzmann[11] emphasized the fact that sclera can be split. Lauber[12] has shown on a cadaver eye that the sclera can be split into two layers but not the cornea, and that at the corneal margin there is a special obstacle to splitting. It is noteworthy that scleral cysts are usually encountered in children; this is also true of spontaneous internal scleral ruptures.

The liquefaction of the sclera in the region of the internal scleral sulcus must be distinguished from the real laceration of the internal lamellae. Intercallary staphyloma may also develop by liquefaction. Such pictures are found in Samuels' *Fuchs Clinical Pathology of the Eye*.[13] Staphyloma due to liquefaction occur in cases of retinoblastoma, as seen in plate 44/5; or in inflammations (for instance due to syphiloma) (plate 26/3). In the case of Vogt-Koyanagi's uveitis in both eyes, enormous intercallary staphyloma developed in a short time (fig. 68).

Also liquefaction and splitting can occur at the same time (fig. 144); an intercallary staphyloma was formed as a result of a leproma of the ciliary body. The granuloma had enormously extended the internal scleral sulcus and, at the same time a split or slit was

formed, reaching into the cornea. A larger slit in the neighboring sclera was filled with granuloma. Also a ledge developed here, the anterior edge of which deviated toward the inside.

Summary

Spontaneous lacerations of the sclera in the region of the internal scleral sulcus due to secondary glaucoma were described. In three cases a retinoblastoma filled the newly formed pocket (figs. 1, 2, and 3); in one case some shreds of dikytoma (fig. 4) were present in a large cavity; and in one case the pathologic alteration was due to a choroidal angioma. In a case of buphthalmos a laceration into the sclera and into the cornea extended from an intercallary staphyloma (fig. 6). Specially remarkable was a similar splitting in a case of epithelial ingrowth (fig. 7). The sclera can also be, without prolonged increase of tension, lacerated in the region of internal scleral sulcus because of septic abscess (fig. 8). The internal laceration of the sclera consists mainly in a splitting and separation of the external lamellae from the inner lamellae of the sclera. At the same time the scleral spur often deviates inward, due to the insertion of the ciliary muscle or due to the strong elastic elements which here surround the circular scleral bundles (fig. 9).

Liquefaction causing intercallary staphyloma can be combined with the splitting of the sclera.

K. Wolfstrasse 14.

References

1. Mathewson: Am. J. Ophth., **15**:139, 1932.
2. Michel: Bull. Soc. ophtal. franç., 1948, pp. 61-63.
3. Fuchs, E.: Arch. f. Ophth., **79**:53, 1912.
4. Stölting: Klin. Monatsbl. f. Augenh., **51**:5, 1913.
5. Richman: Am. J. Ophth., **19**:792, 1936.
6. Dejean and Boudet: Bull. Soc. ophtal. franç., 1952, p. 303.
7. Böck: Ztschr. f. Augenh., **69**:17, 1929.
8. Bergmeister: Arch. f. Augenh., **79**:285, 1911.
9. Fuchs, A.: Ztschr. f. Augenh., **67**:130, 1920.
10. Samuels, B.: Tr. Ophth. Soc. U. Kingdom, **72**:327, 1954.
11. Salzmann: Ztschr. f. Augenh., **92**:275, 1937.
12. Lauber: Arch. f. Ophth., **58**:220, 1940.
13. Samuels, B., and Fuchs, A.: Clinical Pathology of the Eye. New York, Hoeber, 1952.

THE GOLDMANN APPLANATION TONOMETER*

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Goldmann and Goldmann and Schmidt have described a new version of the applanation tonometer, its theory, calibration, and some of its uses.[1-5] These communications have aroused much interest. We have enjoyed the use of a pilot model of the tonometer for the last two years, and we have come to feel that the instrument represents a major advance in clinical tonometry. It is the purpose of this paper to present a practical guide to the use of the instrument developed from the teaching of residents.

The principal objection to identation-type tonometers (Schiøtz, Gradle, and McLean) is that such tonometers do not offer a direct measure of intraocular pressure. The indentation displaces a relatively large volume of fluid; the displaced fluid distends the ocular coats; and an elevation of intraocular pressure results. The scale reading of the tonometer is an index of the resultant elevated pressure (P_t). However, P_t depends not only upon the initial intraocular pressure (P_o), but also upon the resistance of the walls of the eye to distension (scleral rigidity). Friedenwald developed a method for estimating scleral rigidity and P_o with the Schiøtz tonometer on the basis of scale readings obtained with different plunger loads. The inaccuracies of this method are well known. On the other hand, the Goldmann applanation tonometer offers a direct estimate of P_o.

THEORY

The basic theory of applanation tonometers is simply that when a flat surface is pressed against the cornea, the pressure in the eye may be measured by the force applied to the plane and the area of contact between the plane and the cornea.

$$\text{Pressure} = \frac{\text{Force}}{\text{Area}}$$

With the Goldmann applanation tonometer one measures the force required to establish a standard area of contact.

If the area of contact is kept to a small value (diam. 3.06 mm.), the volume of fluid displaced by the flattening process will be small (0.5 mm.^{3} in a cornea of average radius of curvature), and the pressure in the eye will be elevated only slightly; that is, the P_t which is measured will be very close to P_o.

Variations in corneal curvature make no appreciable difference in displaced volume or estimate of pressure.

Goldmann points out that the surface tension of the film causing the plane to adhere to the cornea and the resistance of the cornea to deformation must both be taken into consideration, and he has allowed for these opposing forces in the calibration of the instrument.

THE INSTRUMENT (fig. 1)

A. *The plane surface* is a transparent piece of plastic seven mm. in diameter mounted on the end of a lever. The lever is hinged and can be swung out of the way when the tonometer is not in use.

B. *The device for estimating the correct contact diameter* is adjacent to the plastic plane and consists of a pair of prisms one

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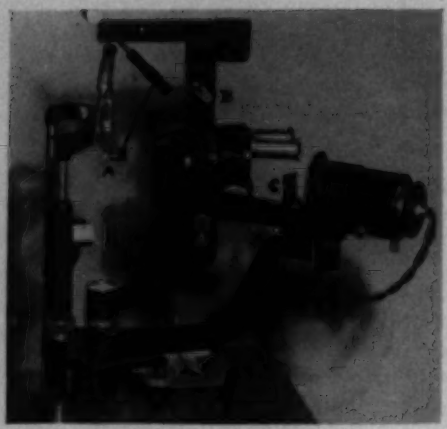

Fig. 1 (Moses). Goldmann applanation tonometer mounted on Haag-Streit slitlamp microscope. (A) Applanation prism. (B) Tension knob. (C) Blue filter.

next to the other with their bases in opposite directions (fig. 2), so that the field seen through the prism pair is displaced, upper half to the left, lower half to the right by a fixed amount (fig. 3).

The edge of the contact area is made visible by instilling a small amount of fluorescein in the tears and viewing in a blue light. The meniscus of tear fluid now appears light green, the rest of the field blue. Of course, the inner edge of the meniscus defines the contact area, and it is this edge which must be matched in the two fields.

C. *The force applied to the prism* is that of a coil spring transmitted through a series of levers. The force is varied by varying the length of the spring. This is accomplished by manually rotating a dial calibrated directly in centimeters of mercury.

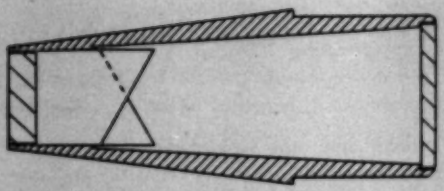

Fig. 2 (Moses). The prism assembly, schematic side view.

The entire tonometer is mounted on the Goldmann (Haag-Streit) slitlamp microscope. The contact area is illuminated by the slitlamp (slit wide open) and is viewed through the right ocular of the microscope at magnification ×20. A blue filter in a hinged mount is rotated in front of the slit.

Use of the Tonometer

The blue filter is swung in front of the slit, the slit is opened maximally, and the light switch is turned to the position for greatest illumination. The slit beam is aimed at the black line on the lateral surface of the tonometer prism.

A drop of anesthetic is instilled into the patient's eye and a wet fluorescein paper is touched to the inside of the lower eyelid. He is then positioned as for microscopy.

The entire assembly is brought toward the eye of the patient until it is seen that the black line moves slightly in the slitbeam. Then, contact with the eye having been established, the semicircle pattern is observed through the right ocular of the microscope. The operator raises or lowers the microscope assembly until the two semicircles are equal in size. He then turns the tension knob first one way and then the other, to assure himself

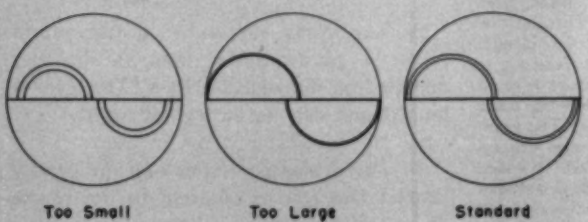

Fig. 3 (Moses). The pattern as seen through the microscope. The arcs are fluorescein-stained tear fluid and are light green against a blue background.

that the pattern can be made "too small" and "too large," and that the instrument is in measuring position (fig. 3).

If the pattern cannot be made "too small," the instrument is so far forward that the lever has been pushed back against the guard spring, and the assembly must be rolled back a little by means of the "joy stick" control until the pattern can be made "too small." If the pattern cannot be made "too large," the instrument is too far away from the eye, and the lever is resting against the forward stop.

When one is assured that the instrument is at the proper height (semicircles equal in size) and in measuring range (semicircles can be made to miss each other and also to overlap), the measurement is then made by turning the tension knob until the semicircles interlock, the inner edge of the upper coinciding with the inner edge of the lower. The setting of the tension knob is observed and the reading $\times 10 =$ I.O.P. in mm. Hg.

Errors in measurement

1. The lids must not touch the prism. Friction between the lid margin and the prism may cause too high an estimate of pressure.

2. Lashes must not be caught between prism and cornea. If they are, they can be observed through the microscope, and the instrument must be drawn away and readjusted.

3. Improper surface tension of the anesthetic. Goldmann and Schmidt emphasize that the surface tension of the tear film influences the reading. However, we have estimated the meniscus attraction force of various solutions and find that the variation is small in clinical terms (table 1).

4. Improper cleaning of the tonometer. Goldmann and Schmidt again state that the surface of the prism has been treated to give it standard wetting characteristics and that it should be cleaned and sterilized with special solutions. We have merely washed our prism with hand soap and water and have found that after numerous such cleanings, the calibration of the tonometer against manometric pressure in a freshly enucleated eye was excellent (fig. 4).

TABLE 1

A. Surface tension

The microscope was removed and a rubber bulb (approximately 8.0 mm. of curvature) was mounted so that the surface would contact the back surface of the tonometer prism. A drop of fluid was introduced between bulb and prism and the measuring knob was rotated until the prism pulled away from the bulb.

Substance	Scale Reading ×10 Required to Break Contact
Saliva	2.0 –2.5
Pontocaine	3.0
Dorsocaine	3.0
Ophthaine	3.0
India ink	3.00–3.75
Distilled water	3.0 –4.0

5. Improper adjustment of height. If the semicircles are unequal, an excess of force will be required to make their inner edges interlock (table 2, fig. 5).

6. Prolonged contact between cornea and prism causes fluorescein staining of the cornea. While it has been easy to obtain clinical pressure measurements without this occurrence, experiments which involve multiple readings in a short space of time have been unsatisfactory due to staining of the corneal epithelium. Prolonged contact also causes "flowing" of the corneal substance and an

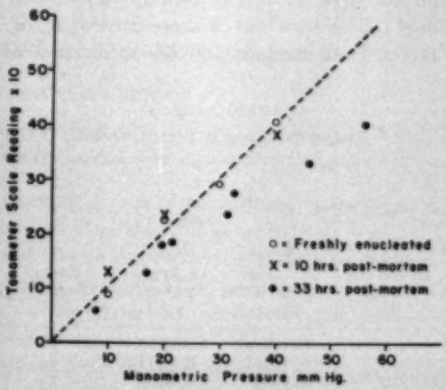

Fig. 4 (Moses). Calibration against enucleated human eyes.

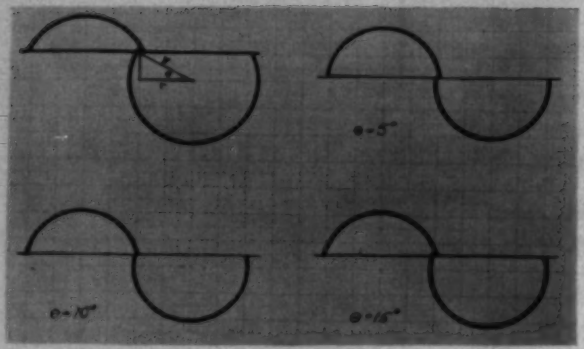

Fig. 5 (Moses) Influence of vertical decentration of the prism on the cornea. Θ = Decentration in degrees. r = Standard radius of applanation area. y = Actual radius of applanation area.

apparent decrease in pressure over a period of minutes.

7. Moderately eccentric positioning on the cornea seems to have little effect on pressure reading.

8. Where there is marked corneal astigmatism, the contact area is elliptical rather than circular. The prism must be rotated so that the dividing line lies at about 45 degrees to the major axis of the ellipse.[5]

9. Corneal edema, scars, bloodstaining, or any condition which thickens or alters the characteristics of the cornea probably gives unreliable results. Thus, calibration against old enucleated eyes is unsatisfactory (fig. 4). The accuracy of measurements in keratoconus is unknown.

10. Animal eyes have not corresponded to human eyes so far as tested (rabbit, cat,[6] dog[6]). The structure of these corneas is different from humans and the calibration of the instrument depends upon a particular structure. However, linear calibration scales can be readily derived for rabbit eyes (fig. 6).

11. Improper placement of the light beam. If the iris is too brightly illuminated, the fluorescein pattern is lost. Merely aim the beam further back on the prism.

12. Goldmann and Schmidt find that the width of the meniscus influences the tonometer reading slightly, the narrower menisci giving more accurate results, the wider menisci causing slightly high pressure estimates.

TABLE 2 (fig. 5)

ERROR OF VERTICAL DECENTRATION

$$y = \frac{r}{\cos \theta}$$

Angle of Decentration θ	Radius of Applanation (cm.)	Area of Applanation cm.²	Excess Area %
0°	0.1530	0.0735	0
5°	0.1536	0.0741	1
10°	0.1554	0.0758	3
15°	0.1584	0.0788	7

LEARNING TO USE THE TONOMETER

One should be able to learn to use the in-

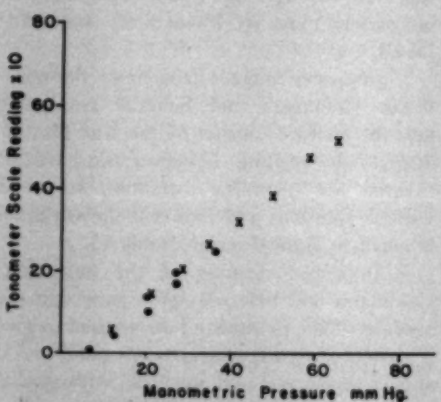

Fig. 6 (Moses). Calibration against enucleated rabbit eyes.

strument in a half hour if one is already familiar with the Goldmann slitlamp microscope. After the learning period, individual measurements should take no more than one minute.

Ruggedness

Our instrument has been in the out-patient clinic, where it has been used by many different operators including all of the postgraduate students and residents. We are certain that it also has been tampered with by several unauthorized persons. It has developed no defect, the calibration on enucleated eyes remaining the same.

Advantages and disadvantages

Advantages

1. Sitting position at same instrument used for slitlamp microscopy.
2. (Most important): Estimates of intraocular pressure are made virtually free of "scleral rigidity."
3. An applanation tonometer reading plus a Schiøtz tonometer reading provide an excellent basis for scleral rigidity estimates.[3]

Disadvantages

1. Pressure estimates are uncertain in corneal edema, opacity, and irregularity.
2. The tonometer cannot be used readily on recumbent patients (especially infants).

Summary

The Goldmann applanation tonometer is a precision instrument for direct estimation of intraocular pressure. Its chief importance lies in the fact that measurements made with the tonometer are virtually free of the errors due to variation in scleral rigidity inherent in indentation tonometer measurements.

The applanation tonometer is sturdily built and simple to use.

The theory of the instrument, its construction, the method of its use, sources of error, and its advantages and disadvantages have been described and discussed.

640 South Kingshighway (10).

References

1. Goldmann, H.: Bull. Soc. franç. ophtal., **67**:474, 1955.
2. ———: On applanation tonometry. Tr. II Conference on Glaucoma. (Edited by F. W. Newell.) New York, Josiah Macy, Jr., Foundation Publications, 1957.
3. Schmidt, T.: Klin. Monatsbl Augenh., **129**:196, 1956.
4. ———: Ophthalmologica, **133**:337, 1957.
5. Goldmann, H., and Schmidt, T.: Ophthalmologica, **134**:221, 1957.
6. Lawrence, C.: Personal communication.

PATIENTS' ACCEPTANCE OF CORNEAL MICROLENSES

A study based on a questionnaire survey

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Until recently the low degree of tolerance to contact lenses has been a deterrent to their prescription. The new microlenses have overcome many of the endless difficulties of fitting, the problems of fluid with proper pH, and the frequency of veiling. It had been my clinical impression that these new lenses gave a much higher percentage of satisfactory wearing time. To measure the truth of this impression, a survey of patients in the San Francisco Bay area fitted with these lenses during the past two and a half years was conducted by mail. The prescriptions came from a large number of doctors but were dispensed by one optical company. An explanatory letter was enclosed with a form to be completed by the patient and a space was provided for his signature. Of 1,300 letters

sent out, 70 were unclaimed. Of the 1,230 delivered, 613, or approximately 50 percent, were answered. This seems like a fairly satisfactory response to such a query.

Of the 613 patients who answered, 553 (90.3 percent) were wearing their lenses, and 60 (9.7 percent) were not wearing their lenses. Of the 553 wearing their lenses, 52 percent were wearing them more than 12 hours a day, and almost 70 percent were wearing them more than eight hours a day. If the week is divided into 14 half days, we find 58 per cent of them were wearing them more than 75 percent of the week's time, and 73 percent more than 50 percent of the week. A few of the people obviously wore the glasses only occasionally or for special events. For example, one woman stated that she wore her glasses three days a year. Only 4.2 percent of them wore their glasses less than four hours a day, and 4.6 percent less than 10 percent of the week.

Of the 9.7 percent of patients not wearing their lenses, about 50 percent had tried to wear them for two months or more, and about 25 percent had tried to wear them for less than two months. In some cases, the answers to the questions were incomplete or omitted and the statistics do not always come out to 100 percent.

The reasons for not wearing the glasses were somewhat varied, nonspecific, and at times unrelated to ocular problems. Of the 613 fitted, approximately five percent were unable to wear their lenses because of irritation, 1.3 percent because of speck blur, 2.2 percent because of problems of insertion or removal technique, 1.6 percent because of losing or breaking the lenses, 0.1 percent because of corneal abrasion, and 0.3 percent for other reasons. Among the "other reasons" one young girl said "got married, didn't need to any more." One man was perfectly happy with his lenses, but he kept losing the tiny things and was embarrassed to have the insurance company replace them.

The very low incidence of corneal abrasion, even if we include those cases listed as irritation, speaks well for the general freedom from trauma by these microlenses. I have observed infrequent instances of mild epithelial stippling unassociated with symptoms of irritation, which have necessitated a reduction in wearing time. However, the cornea seems to develop increased resistance to this irritation, or a more nearly perfect fit eliminates the stippling, so that usually increased wearing time is possible.

The age of patients fitted with lenses is indicated in Table 1. The youngest patient, a girl aged 13 years, is wearing her glasses every day all day. The percentage of rejection seems essentially unrelated to age, although there are mild variations within each division. The high percentage of aged people able to insert and tolerate the lenses is an indication of increased facility of technique and improvement of fitting.

Women outnumbered men about three to one and the men have a slightly higher incidence of rejection. I have observed that men who demonstrate feminine traits are less inclined to wear their corneal lenses successfully. I personally, am extremely tolerant of my own contact lenses since discovering this.

The nature and the amount of the refractive error apparently plays little part in the degree of tolerance. Of the 613 patients fitted, 498, or about 80 percent were myopic, while 17, or about three percent, were hyperopic. This does not include those with aphakia. Three out of the 17 hyperopes, or 18 percent, rejected their lenses, while less than 10 percent of the myopes did. The amount of the myopia was of some importance in predicting rejection.

Among those fitted with lenses and having an error of less than one diopter were only four myopes and they were all wearing their lenses. However, this small number has no statistical significance. Only nine out of 200 myopes, with a refractive error greater than five diopters, gave up wearing corneal lenses. This is a rejection percentage of 4.5 percent, while 13 out of 95, or 15 percent with a

TABLE 1

STATISTICAL BREAKDOWN OF QUESTIONNAIRES

Classification		Number Wearing Plus Number not Wearing	Number not Wearing	Percent of Classification not Wearing	Percent of Total in Classification
Age in years	Less than 20	91	7	7.7	14.8
	20–30	234	26	10.6	38.2
	30–45	183	14	7.7	29.8
	45–60	78	9	11.5	12.7
	More than 60	22	2	9.1	3.5
Sex	Male	163	21	12.9	26.6
	Female	452	37	8.2	73.4
Years worn glasses	Less than 5	51	3	5.9	8.3
	5–10	153	15	9.8	25.0
	More than 10	400	27	6.8	65.3
Percent of life worn glasses	Less than 25	70	6	8.6	11.4
	25–50	94	11	11.7	15.6
	50–75	387	27	7.0	63.2
	More than 75	46	7	15.2	7.8
Reason for contacts	Medical	121	11	9.1	19.8
	Social	492	41	8.4	80.2
Hours per day wearing contacts	Less than 4	26			4.2
	4–8	103			16.8
	8–12	109			17.8
	More than 12	318			52.0
Percent of week wearing contacts	Less than 10	28			4.6
	10–50	72			11.4
	50–75	95			15.5
	More than 75	358			58.0
Time tried contacts if not wearing	Less than 1 mo.		9		1.4
	1–2 mo.		12		2.0
	2 months		32		5.2
Wearing contacts with myopia of (diopters)	Less than 1	4			.6
	1–3	95	13	2	13.3
	3–5	184	18	29	27.2
	More than 5	200	9	14	31.2
Keratoconus		19	2	10.5	3.1
Aphakia	Mono	59	6	10.2	9.6
	Bi	20	2	10.0	3.3
Astigmatism (diopters)	Less than 1	338	18	5.3	55.0
	1–3	128	12	9.9	20.5
	3–5	15	2	13.6	2.4
	More than 5	7			1.1
Corneal scars		8	2	25	1.3
Use	Near	343	15	4.4	56.0
	Far	235	23	9.9	38.4
Reason for not wearing contacts	Irritation		31		5.0
	Spectacle blur		8		1.3
	Technique		14		2.2
	Lost, Broken		10		1.6
	Abrasion		1		.1
	Other		2		.3
Wearing with hyperopia of (diopters)	Less than 1	1			.1
	1–3	5	1	20	.9
	3–5	7	1	14.3	1.1
	More than 5	4	1	25	.7

myopia of between one and three diopters abandoned them. However, the longer a patient had worn regular glasses, the less inclined he was to tolerate corneal lenses. Over 15 percent of those who had worn glasses more than 75 percent of their life abandoned the corneal lenses fitted to their prescription. The amount of astigmatism seemed of some importance, in that as the astigmatism increased from less than one unit up to five, the percentage of tolerance decreased. However, all of the seven patients with astigmatism of more than five diopters, were wearing their lenses.

The 613 persons fitted with lenses were further divided into those who had been fitted for medical reasons and those who had been fitted for social reasons. The medical, 121, or 19.8 percent, and the social, 492, or 80.2 percent, both showed a rejection rate of almost exactly the same, 9.1 percent in the former and 8.4 percent in the latter. The greatest number of medical prescriptions were for aphakia (79) of which 59 (9.6 percent) were monocular, and 20 (3.3 percent) were binocular. They each had about 10 percent rejections. There were 19 (3.1 percent) keratoconus, with two (10.5 percent) not wearing. Lenses were given to eight patients with corneal scars, of whom two were unable to wear them.

In an effort to evaluate the effect of the type of work upon the acceptance of corneal lenses, we divided the group into those who did essentially near work (students, office workers, and so forth) and those essentially using their eyes for distance work (laborers, farmers, housewives, and salesmen). Of 343, or 56 percent, classed as near workers, there were only 4.4 percent nonwearers; whereas, 235 (38.4 percent) classed as far workers had 9.9 percent rejection.

In the specific occupations, students led the list with 22 percent. Of these, approximately nine percent were not wearing their glasses. The next most common group was the housewife, of which there were 19 percent and approximately 10 percent of these were not wearing their glasses. The next most common group were office workers, approximately 14 percent; teachers four percent, of whom approximately eight percent rejected their glasses; executives 4.6 percent, among whom there were no rejections; profesional workers 3.7 percent, among whom there was a rejection rate of approximately 15 percent. Laborers fitted with lenses accounted for 2.3 percent, and their rejection rate was approximately 23 percent.

Summary

1. A questionnaire was received by 1,230 patients asking about the use of their corneal lenses, 613 responded and indicated that 90 percent were wearing and 10 percent not wearing their lenses; 70 percent of those wearing their lenses were wearing them more than eight hours a day.

2. Tolerance seemed to be mildly related to occupation (near workers more tolerant than far workers). Men were slightly less tolerant than women. The greater the astigmatism, the less was the tolerance. Myopes of more than five diopters were more tolerant than myopes of lesser degree. Those who had worn glasses for a long time were less tolerant.

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Acknowledgment

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TRAUMATIC HYPHEMA

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Hyphema from contusions is one of the neglected subjects of ophthalmology. It is, apparently, not until he is confronted with his first complicated case that a younger man becomes aware of the paucity of available information in our standard texts, and the limited number of articles in the journals. This was rather sharply pointed out by Thygeson and Beard[1] in 1952.

In the latest authoritative book (Duke-Elder, Volume IV[2]) three pages and three microphotographs are devoted to the entire subject, including complications and treatment. In the more advanced texts, Spaeth[3] dismisses the subject with two sentences, and Callahan[4] covers the subject in two paragraphs, including complications and treatment. The standard medical school texts, May,[5] and Gifford,[6] dismiss the subject with little more than a mention. It is hardly any wonder that the general practitioners continue to treat cases of this nature without being aware of the severe complications that may arise.

With such scant treatment, and the rather off-handed dismissal of hyphema, eyes continue to lose usable vision and a few have to be enucleated, regardless of who supervises the treatment, or what methods of treatment are employed. It would, therefore, seem that further clinical investigation, with particular emphasis on the statistical aspects of complications, is in order.

Pathologic specimens are difficult to obtain until late disorganization predominates the picture. The opportunity is thereby denied us to find and identify the basic cause of primary and secondary hyphema.

Only two reports of experimental research have been published. Kilgore,[7] working with contusion of the eyes in animals, indicates that ciliary body tears are the most frequent basic pathologic lesion. Detachment and tears of the ciliary body occurred about the middle, and may be in the area of damage, or on the opposite side. He felt that this lesion explained the hemorrhages better than tears at the iris base, and that secondary hemorrhage could be explained by seepage from this site. Iridodialysis may, or may not, be present.

Sinsky, et al.,[8] studied the question of whether red blood cells get out of the anterior chamber as whole red cells or are washed out in the hemolyzed state. By using whole and hemolyzed cells that were tagged with P^{32}, in rabbits, they found that: (1) whole red cells can get out of the anterior chamber without undergoing hemolysis first and, apparently, get out relatively easily and (2) hemolyzed blood left the chamber readily and most was washed out of the peripheral blood by the kidneys.

Thygeson,[1] in two eyes obtained by enucleation, found ciliary body damage was the predominant lesion in each.

Duke-Elder,[2] on the other hand, has microphotographs of an eye in which the basic lesion is an iridodialysis, with ultimate disorganization of the eye.

Traumatic hyphema is the accumulation of blood in the anterior chamber, as a result of contusion of the eye. The term "concussion" is applied to the reversible effects of a blunt injury. "Contusion" is the next degree of severity in which there is a dissolution of some tissues, although the end-result may be reversible; that is, complete recovery of the eye.

Fifty-six cases of hyphema are presented in this series.

CAUSES

Table 1 presents the wide variety of causative agents that have come under observation. BB shot (10 cases) and thrown objects of 11 various types (15 cases) account for nearly half of all causes; 22 of these 25

TABLE 1

OBJECTS CAUSING CONTUSION

Objects	Number of Contusions
BB shot	10
Rock, thrown	4)
Clod of dirt, thrown	2)
Berry, thrown	1)
Baseball, thrown	1)
Bat, thrown	1)
Football, thrown	1)15
Shingle, thrown	1)
Hardy candy, thrown	1)
Corn cob, thrown	1)
Carrot, thrown	1)
Iron rod, thrown	1)
Objects struck with hammer	4
Fists	3
Firecracker explosion	3
Rope, whipped	3
Fluid under high pressure	2
Blackjack or similar object	2
Hit by bush	2
Arrow	2
Rubber band	1
Sling shot (band)	1
Spitball from rubber band	1
Beer can	1
Steel spring	1
Stick	1
Finger in eye	1
Falling object (paper)	1
Kicked in eye	1
Automobile accident	1
TOTAL	56

occurred in patients under the age of 15 years. Fists and thrown objects accounted for three each in the older age group. Evidently this younger group is more exposed to these types of injuries. The balance were caused by isolated agents. The eyes involved were fairly evenly distributed; 25 right, and 31 left. No bilateral cases were seen. Two patients were Latin-American, and six were Negroes.

AGE

Table 2 lists the age incident. There is a rather unusual gap between the ages of 15 and 26 years that is not explainable. The majority of cases, 36 of 56, occurred between the ages of four and 15 years, inclusive, or 64.3 percent; this fact has been noted by other authors. The ages of the remaining patients range from 26 to 58 years. Six of the younger group were females, ages 4, 9, 11, 13, 15 years, and one was 58 years of age. All others in the entire group were males.

TABLE 2

AGE INCIDENT

Age (yr.)	Number of Cases	Age (yr.)	Number of Cases
4	3	26	1
5	2	27	2
6	3	28	2
7	3	29	2
8	2	30	1
9	4	31	1
10	3	34	3
11	5	35	1
12	5	37	1
13	4	39	2
15	2	41	1
	(36)	45	1
		53	1
		58	1
			(20)
TOTAL CASES			56

ASSOCIATED LESIONS

Table 3 lists 40 injuries that could be diagnosed in addition to the basic contusion that all sustained. Associated lesions frequently could not be diagnosed until after the hyphema absorbed. In addition there were 32 eyes that showed more than transient pupillary disturbances, although some of them did not become obvious until some days after the original injury. In all cases seen shortly after injury the pupil was miotic, due to spasm. In those eyes with a lasting disturb-

TABLE 3

ASSOCIATED LESIONS AT TIME OF INJURY

Type of Lesion (some multiple)	Number of Lesions
Central edema (retina)	10
Lid injuries	10
Iridodialysis	4
Corneal laceration (nonpenetrating)	5
Conjunctival laceration	4
Chemical burn	1
Vossius ring	2
Choroid rupture	2
Macula hemorrhage	1
Lens damage (cataract and partial absorption	1
TOTAL	40

ance, the pupil dilated, frequently eccentrically to a varying degree probably due to paresis of the sphincter, and either did not respond, or responded sluggishly, to light. Whether this is due to muscle or nerve damage has not been determined. The time of onset was variable. Most of these eyes also had some paresis of accommodation that lasted less time than the mydriasis. Only eight of the pupillary disturbances returned to normal in a few weeks' time. The balance (24 eyes) persisted for two months or more. Many undoubtedly will be permanent. Sphincter rupture or iridodialysis were not diagnosed in these cases. Children predominated 23 to nine.

Those eyes that developed vitreous hemorrhages (six cases) retained a widely dilated pupil that was extremely resistant to all miotics including di-isopropyl fluorophosphate (DFP). This agent was used only in very late stages because of the possibility of severe ciliary spasm causing further hemorrhage. In four instances where the vitreous hemorrhage absorbed fairly rapidly the pupil became active to light, was only semidilated or normal.

Complications

Secondary hemorrhage and glaucoma are the most serious and severe complications of hyphema. Secondary hemorrhage may be either single or recurrent, and in either small or large amount. There is no pattern that can be described. Frequently several different color layers or areas of clotted blood can be noted in the anterior chamber. Primary hemorrhages usually remain unclotted due to aqueous dilution. Secondary hemorrhages, depending on their severity, are often clotted, and usually nearer the iris than the cornea; although the whole chamber may be filled, and no iris remain visible.

Secondary hemorrhages occur between the second and fifth day after injury. Table 4 lists the day-to-day distribution in this series; most being concentrated on the second, third, and fourth days.

TABLE 4

Time of second hemorrhage

Day After Injury	Number of Secondary Hemorrhages
1	0
2	5
3	9
4	8
5	1
Total	23*

* In 17 eyes
6 eyes had 2 recurrences:
3 on 2nd and 3rd days
1 on 2nd and 4th days
1 on 3rd and 4th days
1 on 3rd and 5th days.
2 eyes seen on the 7th day are itemized as the 2nd and 3rd day by history.
1 eye was reinjured and had a second hemorrhage on the 4th day.

Secondary glaucoma (intraocular pressure over 25 mm. Hg (Schiøtz, newer scales) occurred in cases of secondary hemorrhage, and then only when hemorrhage was severe and/or recurrent. One exception should be noted: In one girl (Negro), aged 13 years, who had a severe primary hemorrhage, without recurrence, the blood formed a partial clot which had not absorbed by the fifth day and the intraocular pressure began to rise above 25 mm. Hg (Schiøtz). Medical treatment failed to lower the pressure. Unfortunately the mother refused surgical intervention and removed the patient from supervision and I am unable to furnish data regarding the ultimate result. Most contused eyes after a transient hypertensive period become hypotensive. Small secondary hemorrhages can occur without raising the intraocular pressure to a dangerous degree (seven cases). These cases usually absorb satisfactorily, and can be controlled medically.

Table 5 shows the age distribution of complications. There were 17 of 56 eyes (30 percent) that developed secondary hemorrhages: seven of these recurrences had no significant glaucoma, whereas 10 developed secondary glaucoma. Fourteen of these 17 cases of recurrent hemorrhage, and nine of the 10 cases of glaucoma, were in the four-

TABLE 5

AGE DISTRIBUTION OF COMPLICATIONS

Age (yr.)	Total in Age Group	Secondary Hemorrhage Without Significant Glaucoma	Secondary Hemorrhage With Glaucoma	Vitreous Hemorrhage With Glaucoma	Vitreous Hemorrhage Without Glaucoma
4	1		1		
6	1		1*	1*	
7	2	1*	1		1*
8	1		1§		
11	4	2	2*	2*	
12	4	1	3		
15	1	1†			1†
26	1	1			
29	1		1‡	1‡	
45	1	1			
TOTALS	17	7	10	4	2

* Same eyes in each of 4 cases.
† Vitreous and pre-retinal hemorrhage at time of original injury.
‡ Probable, same eye.
§ Postoperative recurrence with "8" ball cornea. Reoperated 4th day postoperative.

to 15-year age group. Six eyes, all in the younger age group, had recurrent hemorrhages, on two separate days, subsequent to the original hyphema.

Another complication noted was vitreous hemorrhage. This was found in those instances that had secondary hemorrhages, or was not noted in the uncomplicated cases due to the cloudy anterior chamber. This latter is doubtful since no cases were found even after the hyphema had absorbed, and it is questionable that a significant vitreous hemorrhage would absorb as rapidly as an anterior chamber hemorrhage.

Vitreous hemorrhages were found in six instances, four with glaucoma (table 5). The amount and severity varied widely, since four of these six cases cleared well.

Contrary to other authors, only three cases of blood staining of the cornea and lens were noted. Two of these eyes had severe recurrent hemorrhages and glaucoma, and one had multiple injuries. One other case was noted in an eye that went on to complete degeneration.

A late complication in eight cases was the appearance of a cataract. In two instances that had vitreous hemorrhages the cataract developed as a posterior capsular and subcapsular opacity. Two eyes that had prolonged contact with clotted chamber blood developed epithelial proliferation and some isolated cortical opacities. Two other eyes went on to develop complete cortical cataracts and one additional eye developed a complete cortical cataract with absorption of the superior temporal portion of the lens. One eye in a 58-year-old woman had cataract changes present in both eyes (senile type) but the eye with hyphema became progressively more dense while the uninjured eye has remained stationary.

One case of persistent mild iridocyclitis resulted, although an active focus of infection was present in the tonsils of the child. The parents refused tonsillectomy. The ultimate outcome was favorable after a period of several additional weeks. One other eye with iridocyclitis occurred in a neglected case which was not seen until the 17th day.

One unusual reaction should be noted. A 15-year-old boy, first seen on the seventh day, had been given tetanus antitoxin at the time of injury. On the 10th day he developed a typical urticarial reaction. Concomitant with this he had a moderate increase in his macular edema, and an increase in the vitreous haze. Both cleared to their previous level in a few days' time.

VISUAL RESULTS

Table 6 shows the visual result in 48 of

TABLE 6

VISUAL RESULTS

Vision (Snellen) (With glasses if required)	Primary Hemorrhage Only	Secondary Hemorrhage		
		Without Glaucoma	With Glaucoma Medical	With Glaucoma Surgical
20/20	24	5	1	1
20/30	2		1	
20/40	1*			
20/50	2			1
20/70 or Less	3†			
Light perception	2			4
Enucleation advised				1
Undetermined	5(3‡, 2§)	2§	1§	
(56) TOTALS	39	7	3	7

* Corneal scar from superficial laceration.
† Not a final determination.
‡ Too young to determine accurately.
§ Unable to follow patient.

the 56 cases (eight undetermined) in four categories. As may be expected, the best visual results were obtained from the uncomplicated cases. Of seven cases with a secondary hemorrhage, but without glaucoma, five recovered 20/20 (Snellen) vision, and the vision was not determined in two cases. In three cases where the glaucoma was mild, and controlled by miotics alone, the visual result was good in two. In five of seven instances that had some surgical procedure, the visual results were poor; one case recovered 20/20, and another 20/50's vision. Two of these poor results had known vitreous hemorrhages, and a third may have had a vitreous hemorrhage, an occluded pupil preventing a positive diagnosis. This was the only case in which enucleation was advised, although refused. The eye was atrophic and painful in a 29-year-old Latin-American, who had had an industrial accident.

Blood-staining of the cornea did not account for severe visual loss in any case as a sole cause, but was incidental in three cases, two with poor visual results, and one with 20/50 vision.

TREATMENT

The therapy of hyphema is unsettled in many respects. There are enough eyes that recover completely, and without complications when nothing is done, that one could imagine a valid argument, "do nothing unless complications arise." Since the complications are so frequent (30 percent) and the visual results so poor in a high percentage of these cases, I feel that treatment, even if only expectant or prophylactic, should be instituted.

Bedrest, nonuse of eyes, and sedation as indicated, particularly in children, are by far the most important measures. This routine should be continued for at least five days until the danger of a secondary hemorrhage has passed. In the uncomplicated cases the hyphema is usually absorbed by this time. A slow resumption of activities after the fifth day is permitted. It is more difficult to control adults on this regime than children.

Based on what is known of the basic pathology, cycloplegics were used for almost all patients, usually as five-percent homatropine two or three times a day. Atropine was avoided. Miotics as advocated by Rychener[9] were tried in a few early uncomplicated cases, but the patients complained of more pain than when under cycloplegics, and their use was discontinued except in those eyes that developed glaucoma. The absorption rate of blood from the anterior chamber may be more rapid with miotics, due to greater iris exposure, than with cycloplegics, but the number of complications seemed to be about

the same. Iridodialysis contraindicates all miotics.

Synthetic vitamin K, rutin, ascorbic acid, and calcium in different forms were all used either alone, or in various combinations.

Laboratory studies of bleeding and clotting times, platelet counts, and so forth, were done, but none of these showed any abnormality of the blood either before or after the above measures. From a theoretic viewpoint all the above agents have a definite place in control of bleeding mechanism or capillary responses, and their use is, therefore, desirable.

Ice bags, binocular bandages, and pinhole goggles to immobilize the eyes further were tried but seemed to upset the patients, and make them more apprehensive. Their use has been discontinued. Heat is contraindicated due to vasodilatation effect.

Secondary hemorrhage, with or without glaucoma, calls for the immediate use of miotics, even if the possibility of further hemorrhage is increased. The filtration angle must be opened in an attempt to prevent a rise in pressure, or to control pressure if already elevated. Pilocarpine (two percent) is preferred every three or four hours. At this time it is rare that one can see the pupil more than vaguely through the cloudy anterior chamber. Other miotics may be added if the miosis seems to be inadequate or the pressure is rising.

Diamox, as advocated by Becker,[10] can be used. On a basis of the decrease in intraocular pressure as reported, Diamox may well prove of great value, especially in mild secondary hemorrhages with minimal increased pressures. The amount of Diamox required is relatively more in a child than would be expected, if dosage is calculated on age and weight alone.

These measures frequently control the milder cases.

If the intraocular pressure continues to rise, or is not controlled within a matter of hours by these more conservative agents, paracentesis is indicated. Early paracentesis before the blood is solidly clotted allows adequate drainage without having to remove the small clots. This can be repeated as often as indicated. If the chamber is full and the secondary blood is clotted, or if the patient is first seen in this condition, irrigation through a small keratome incision at the limbus, or through and through irrigation from a slant incision, can be done. Blind manipulation in the filled chamber may produce more damage than benefit. It is better to enlarge the incision with scissors and attempt removing the clot under direct observation if necessary. The cornea can be sutured on completion and air instilled, or a beveled incision in clear cornea can be used for air instillation.

Wilson, et al.[11] have advocated the use of early paracentesis and air injection in all cases of primary hyphema. They report extremely effective results with a low incidence of complications. I have not tried their early routine use of air, but have used it with equivocal success in secondary hemorrhage cases. Perhaps the use of an air bubble in the very early stages of secondary hemorrhages, whether the pressure is elevated or not, or is controlled by miotics and/or Diamox, would be more beneficial. It is certainly worthy of further investigation.

In one severe case where a keratome and scissors opening was used, irrigation and forceps failed to remove the clot without probable severe damage to the iris. Varidase, as advocated by Jukofsky,[12] was instilled in the chamber after closure. That is the eye in the above tables for which enucleation was advised. Since that time O'Rourke[13] in a controlled experimental study came to the conclusion that there was slight reduction in absorption time of induced hyphema when the chambers were subsequently injected with streptokinase. A further reduction was noted if there was subsequent aspiration. Smillie[14] using a low concentration of streptokinase without secondary aspiration found no significant difference in the absorption rate. He also calls attention to the toxic sequelae that may result. Apparently this ther-

apy has no place in the treatment of human eyes.

Another thought on therapy was offered by Hopen, et al.[15] with the use of intramuscular Trypsin. They found five of six cases of postoperative hyphema benefited. There is no report concerning primary hyphema. I have been unable to substantiate this form of therapy.

Keeney and Zaki[16] using intramuscular and subconjunctival Trypsin experimentally in guinea pigs failed to produce any appreciable effect on the absorption of blood.

The only other class of drugs that has proven beneficial in the late stages are the corticosteroids. According to Benedict and Hollenhorst,[17] cortisone decreases the absorption of blood in experimentally produced lesions, and should therefore not be used while blood remains in the chamber. I have employed these drugs after the fourth day to quiet the residual iridocyclitis. In a few instances, where indicated by corneal damage, or chemical burns with hyphema, I have used them from the start. No appreciable difference in blood absorption was noted.

Discussion

I am aware that misleading conclusions may be drawn from the statistics of this series. There is no way of knowing how many cases of primary hemorrhage occurred and were treated by the same practitioners who referred the other cases. Eight of the complicated cases were not seen until secondary hemorrhage had occurred and included four of the seven surgical cases. The other nine developed secondary hemorrhage while under treatment, three ultimately requiring surgical care. The fact that 30 percent of all the cases developed second hemorrhages is slightly higher than the reported statistics of Thygeson and Beard,[1] 26 percent (13 of 34), and well above Wilson, et al.,[11] figure of seven percent (two of 27 percent). Duke-Elder gives no statistics.

The poor visual result (20/70, Snellen, or less) in 10 out of 48 (eight undetermined) cases, 20 percent, is serious enough; but when analyzed as five out of 14 (three undetermined), or 35 percent of the complicated cases, the significance increases, and increases even further with five out of seven surgically treated cases resulting in poor vision. Any one eye would have a 30 percent chance of developing complications, and then one out of three will have a poor visual result.

Duke-Elder[2] states: "while primary hemorrhages are most common in children, the secondary type is seen most frequently in older patients."

Statistics of this series indicate the opposite situation. Children are exposed to more minor injuries, and therefore the high occurrence of primary hemorrhage is explainable. However, 14 of 36 (38.8 percent) developed secondary hemorrhages as compared to three of 20 (15 percent) in the older age group. Using the 15-year-level, Thygeson and Beard would have 20 of 34 cases below the age of 15 years, or 59 percent, and nine of 13 (69 percent) of these with secondary hemorrhages as compared to 31 percent in the other 14 or 41 percent of their cases.

Obviously, the statistics in this small series of cases are no more than an indicator but one pertinent fact stands out: Hyphema in a child is far more serious than in an adult.

In reviewing this series I was impressed by the fact that where the original injury was more severe the likelihood of secondary hemorrhage was decreased, and the more minor the injury the greater the possibility of complications other than the associated lesions. A ricochet BB shot should certainly have less force per unit than a blackjack, fist, whipped rope, or fluid under high pressure. There is no obvious explanation for this observation. This observation in three or four instances induced me to permit adults to remain at home, but not completely at bedrest. I would not advocate this procedure for a child.

There is some doubt about the relationship of secondary hemorrhages, vitreous hemorrhages, and glaucoma. We know that secon-

dary hemorrhage usually precedes both of the other complications. Glaucoma that is easily controlled by miotics is usually seen without vitreous hemorrhage, or at the most a very small hemorrhage. Also, in a mild secondary hemorrhage, a minimal vitreous hemorrhage can occur without glaucoma. Where the glaucoma becomes severe, and extremely difficult to control, severe vitreous hemorrhage is usually present. The end-results of these cases are the poorest recorded.

It is possible that a rapid decompression of a high intraocular pressure could induce a vitreous hemorrhage. These are the reasons for thinking along the line that apparently guided Wilson, et al.,[11] although they do not stress vitreous hemorrhage, to early paracentesis before the pressure is too elevated, and instilling air as a flexible cushion in the chamber to maintain a relative normal pressure, rather than leave a period of extreme hypotension to be followed by elevation.

I am in full agreement with the opinions of Thygeson and Beard,[1] and Rychener,[9] that once a secondary hemorrhage develops the prognosis shifts from good to bad, and if glaucoma develops the prognosis becomes even worse.

Summary and Conclusions

1. Traumatic hyphema has been generally ignored in the current texts. Literature has been scant until the past few years. Statistics are almost nonexistent.

2. Analysis of 56 cases with respect to cause, age, time, and frequency of secondary hemorrhage and glaucoma, and visual result has been presented.

3. The importance of secondary hemorrhage and glaucoma on visual result is stressed. The seriousness of the prognosis is indicated by the visual outcome.

4. The increased seriousness of hyphema in children as compared to adults is indicated, statistically.

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References

1. Thygeson, P., and Beard, C.: Observations on traumatic hyphema. Am. J. Ophth., **35**:977 (July) 1952.

2. Duke-Elder, W. S.: Textbook of Ophthalmology. St. Louis, Mosby, 1954, v. 6, p. 5776, and 1940, v. 3, p. 2109.

3. Spaeth, E. B.: Principles and Practice of Ophthalmic Surgery. Philadelphia, Lea, 1954, p. 535.

4. Callahan, A.: Surgery of the Eye: Injuries. Springfield, Ill., Thomas, 1950, p. 20.

5. May, C. H.: Manual of the Diseases of the Eye. New York, Wood, 1934, pp. 179, 188.

6. Gifford, S. R.: Textbook of Ophthalmology. Philadelphia, Saunders, 1945, p. 367.

7. Kilgore, G. L.: An experimental study of iridodialysis. Tr. Am. Ophth. Soc., **40**:516, 1942.

8. Sinskey, R. M., Krichesky, A., and Hendrickson, R.: Experimental hyphema in rabbits. Am. J. Ophth., **43**:292 (Feb.) 1957.

9. Rychener, R. O.: The management of traumatic hyphema. J.A.M.A., **125**:763, 1944.

10. Becker, B.: Diamox in glaucoma; Am. J. Ophth., **37**:13 (Jan.) 1954.

11. Wilson, J. M., McKee, T. P., Campbell, E. M., and Miller G. E.: Air injection in the treatment of traumatic hyphema. Am. J. Ophth., **37**:409 (Mar.) 1954.

12. Jukofsky, S. L.: A new technique in the treatment of hyphema: A preliminary report. Am. J. Ophth., **34**:1692 (Dec.) 1951.

13. O'Rourke, J. F.: An evaluation of intraocular streptokinase. Am. J. Ophth., **39**:119-139 (Feb., Pt. II) 1955.

14. Smillie, J. W.: Effect of streptokinase on simulated hyphema: With a study of its toxicity to anterior chambers of rabbits. Am. J. Ophth., **37**:911-917 (June) 1954.

15. Hopen, J. M., and Campagna, F. N.: Use of intramuscular trypsin. Am. J. Ophth., **40**:209 (Aug.) 1955.

16. Keeney, A. H., and Zaki, H. A.: The role of Trypsin in experimentally induced hyphema. Am. J. Ophth., **43**:275 (Feb.) 1957.

17. Benedict, W. H., and Hollenhorst, R. W.: Cortisone and blood absorption. Am. J. Ophth., **36**:347 (Feb.) 1953.

NOTES, CASES, INSTRUMENTS

CICATRICIAL OCULAR PEMPHIGUS*

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Since the time when every bullous outbreak was called pemphigus, a great deal of progress has been made regarding the bullae of pemphigus. In the study of these lesions, the different schools went different ways, and the first enthusiasms were followed by many descriptions of the appearance of differential morphologic or etiopathogenic data which would allow a classification of the multiple pemphigus and pemphygoid types. Among the criteria adopted for the differentiation of these types, due to their importance, emphasis should be placed on the evident infectious participation (Streptococcus, Bacillus pyocyaneus, Triponema pallidum, and so forth) and on the hereditary predisposition of the tissues to the formation of blisters (hereditäre Neigung zur Blasenbildung).

As Rabelo and his colleagues state, however, there remain eosinophilia (which is not always connected with allergic or parasitic conditions), Nikolski's sign (which constitutes chiefly a sign of intense exudative dermatitis), the halogen test (which is another exudative stigma) and the histologic image, all of which are linked to the study of the bullae of the pemphigus group, in spite of the fact that they are not differential characteristics.

Specially based on the aforementioned characteristics multiple nosologic entities were described which resemble true pemphigus, which can be differentiated by an essentially negative character—that of showing itself to us in the form of an etiopathogenic enigma. However, little by little this enigma is being penetrated, for not only is the important part played by infection in bullous pemphigus coming to light, but also authentic types of transmission between pemphigus and nonpemphigus bullae are being described.

Cicatricial ocular pemphigus presents difficulties regarding its nosologic identification. Some authors believe that it should be considered a special and independent form, since it is well defined and individualized by the presence of blisters and characteristic residual scars that join the palpebral conjunctiva to the bulbar conjunctiva (symblepharon).

Other authors, taking into account the fact that the lesions of cicatricial ocular pemphigus can compromise, together with the conjunctival mucosa, other mucosa, and even the skin, at the same time or at a different period, believe that this manifestation of bullae should be considered true pemphigus.

Fuchs' case (Ramel) is especially interesting in that the pemphigus bullae remained for a period of seven years localized exclusively on the buccal mucosa, where they caused important cicatricial alterations, later becoming manifest on the skin and, five years later, appearing on the conjunctiva where they caused complete blindness due to cicatricial complications. However, no less interesting is the case I shall present, as the appearance of the bullae in early childhood, their exclusive localization on the conjunctival mucosas for over 80 years, their extreme benignity, are much more similar to hereditary bullous epidermolysis.

The following case, as far as is known, is the first case of cicatricial ocular pemphigus in the Brazilian literature.

CASE HISTORY

M. F., aged 86 years, a Portuguese farmer, was sent to our out-patient department by the Department of Ophthalmology of the Hospital Matarazzo. He was examined on March 10, 1950.

Heredity. His parents were not consanguineous. The patient could not tell whether any of his rela-

* Presented before the Department of Dermatology and Syphilology of the Associação Paulista de Medicina, April 11, 1950, and September 11, 1956.

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tives had bullous affections of the ocular conjunctivas or skin. He had four brothers who died at an early age.

Personal. He could not state whether he had had the usual children's diseases. From childhood until 36 years of age, at which time he came to Brazil, he had frequent attacks of a violent type of febrile influenza with tracheobronchial involvement. At 20 years of age he had pneumonia. Since coming to Brazil, he has had no more attacks of this influenza and his general health has improved considerably. He had seven children, five of whom died in early infancy of unknown causes. About two years ago he had a slight vesiculous eczema, with preferential localization on the legs. This eczema rapidly healed with adequate topical therapy.

History of present disease. Since childhood, he has suffered from irritation and blisters in the eyes and would not permit his face to be washed for fear his eyelids would be touched. However, this affection improved considerably (smaller blisters, less pronounced inflammatory phenomena) after he came to Brazil. He denied having had blisters on other mucosa or on the skin.

Examination. He was a well-built individual of medium height, not showing anything worthy of note in the respiratory, circulatory, digestive, genital, and nervous systems. Ganglia normal in the regions accessible to palpation. Pupils reacting well to light and to accommodation. Nails normal. Regarding integument we noted congestion and moderate parakeratosis of the legs, remains of papulovesiculous crustaceous eczema, and a patch of seborrheic eczema.

There was marked inflammatory cicatricial alteration of the conjunctivas and especially of the palpebral margins (greater on the lower lids) which were very much thickened and completely without cilia. At the time the photograph of the patient (fig. 1) was taken, a large blister took up the entire external third of the lower left eyelid. In Figure 2 are shown a symblepharon and a blister in the external third of the right eye. I should especially like to call attention to the satisfactory visual condition of the patient who can still get about the town without a guide.

Ophthalmologic examination (March 18, 1950). LEFT EYE. Upper lid: cicatricial retraction and pronounced thickening of the free lid margin; absence of line of implantation of cilia; presence of a blister with transparent and aqueous contents, occupying the whole of the nasal third; decreased and partial closure of the upper cul-de-sac. Lower lid: retraction of the middle third (cicatricial coloboma); absence of line of implantation of cilia; thickening of the free margin with a decrease of the palpebral fissure; presence of a blister with the same characteristics of the others in the middle third of the lid. Ample symblepharon occupying the entire inferotemporal half of the bulbar conjunctiva.

RIGHT EYE. Upper lid: cicatricial retraction of the free margin in the middle third (cicatricial coloboma); presence of two blisters with transparent contents and of different sizes; absence of the line

Fig. 1 (Bianco). Blister occupying the entire external third of the lower left eyelid.

of implantation of the cilia; total madarosis; partial closure of the fundus of the upper cul-de-sac; ample and complete arcus senilis; absence of neoformed vessels on the limbus. Lower lid: pronounced thickening of the free margin and absence of the line of implantation of cilia; two blisters with aqueous and transparent contents in the temporal third; absence of lower cul-de-sac; in the middle third the lid is joined to the bulbar conjunctiva by a symblepharon. Cornea with ample arcus senilis in the lower half and slight infiltration of neoformed vessels in the upper limbus. Iris and crystalline lens unaltered.

Visual acuity: 1/4 in the right eye and 1/10 in the left eye (Dr. José Lucas de Souza).

Otolaryngologic examination. Examination of the mucosas of the nose, mouth, and throat did not show the existence of blisters or residual scars. Examination of the sinuses showed nothing abnormal (Dr. Daniel Lopes).

Complementary tests. Wassermann: Repeated twice and performed with three antigens, negative. Hemogram: erythrocytes 4,690,000 per mm.^{3}; hemoglobin 93.7 percent, globular value 1.11; leukocytes 10,900 per mm.^{3} (neutrophils 55 percent, 5 percent being rods and 50 percent segmented; eosinophils four percent; monocytes 11 percent; lymphocytes 30 percent, 15 percent being leukocytoid and 15 percent typical). Schilling's index of deviation of the neutrophils 0.100 (normal 0.003). Velez' index 1/10, II/8, III/62, IV/20, V/O. Plasmatic chlorum 300 mg./100 ml. (normal 380 mg./100 ml.). Plasmatic sodium chloride 494 mg./100 ml. (normal 600 mg./100 ml.). Globular chlorum 200 mg./100 ml. (normal 180 mg./100 ml.). Globular sodium chloride 329 mg./100 ml. (normal 300 mg./100 ml.). Globules/plasma: 1/1.5 (normal 1/2). Hemosedimentation: after 30 min., two mm.; after 60 min., eight mm.; after 120 min., 20 mm.; after 24 hr., 72 mm. Cytologic examination of the liquid from a blister on the lower lid; some degenerated epithelial cells; frequent leukocytes; some polymorphonuclear neutrophils; some polymorphonuclear eosinophils. Mantoux: negative. Examinations of urine and stools nothing of note. Nikolski's test: cutaneous autoinoculation with the liquid from a blister, negative. Percutaneous test: repeated three times with a 50 percent ointment of potassium iodide, negative.

Treatment. Only four applications of radium

Fig. 2 (Bianco). This drawing shows a symblepharon and a blister in the external third of the right eye.

therapy of 50 r at intervals of five days were given in April, 1950, and the therapeutic action of these was surprising as the blisters improved considerably.

The patient was seen again on September 11, 1956: general health good, absence of blisters, and slight improvement of the inflammatory phenomena in the conjunctivas. However, the cicatricial phenomena remained unaltered.

This case is especially interesting because it shows the possibility of an exclusive predisposition of the conjunctivas toward cicatricial bullae, an exclusive predisposition which seems to me to be more than evident (as the affection had lasted for over 80 years) and which occurs with the characteristics of a true "locus minoris resistentiae" of the conjunctival mucosa.

Conclusions

Basing my evidence especially on my own observations, I think it possible that there is an exclusive predisposition of the conjunctivas for the manifestations of cicatricial pemphigus of the bullous epidermolysis type. It also appears that, in a general way, this predisposition, hereditary or not, can become manifest after many years. On the other hand, it has been demonstrated that pemphigus vulgaris can take on the aspect of cicatricial pemphigus, if, when localized in the conjunctivas, the conjunctival mucosas have a predisposition for proliferative cicatricial formations. As Brocq states, it is the terrain that contributes essentially to the symptomatology of the pemphigus and pemphigoid pictures.

It may thus be concluded that there are two forms of cicatricial ocular pemphigus, one less serious and identified with bullous epidermolysis, the other very serious due to the conjunctival localization of pemphigus vulgaris.

Before finishing I should like to comment on the following anamnestic data: since our patient took up residence in Brazil he obtained, without special treatment, a marked improvement of his pemphigus affection and his general state of health, not having had any more severe attacks of influenza to which he had been subjected in Portugal. This improvement in the pemphigoid condition could be interpreted as a result of the better general state of health which our patient enjoyed in Brazil.

Without wishing to attribute the only cause of cicatricial pemphigus to the germs of influenza, to Radaeli's bacillus as the cause of pemphigus vulgaris, and so forth, attention should, however, be called to the frequent aggravating part played by "new infections," or "de sortie," upon multiple diseases. In effect one disease can attract or worsen another disease as Marie affirms, expressing himself better than Milian when describing these indisputable clinical facts. To me, it seems not improbable that the present patient had his bullous affection aggravated by the influenza infection. I also believe that a certain predisposition to the bullae of pemphigus may remain latent, without clinical manifestation throughout the entire lifetime of an individual. This predisposition can condition one or another of the forms of pemphigus if, during the individual's life time, special infectious phenomena occur which act pathologically upon this predisposition.

Summary

A case of conjunctival pemphigus, in a man, aged 86 years, is presented. Although the patient has suffered from this disease since childhood his vision is still satisfactory. It is concluded that there are two forms of conjunctival pemphigus: the first type, less severe, can be described as a bullous epidermolysis; the second form, more severe, is caused by the localization of pemphigus vulgaris in the conjunctiva.

Rua Benjamin Constant 171 (9° andar.).

I wish to thank Prof. Humberto Cerruti in whose laboratory the tests referred to in the case history were carried out, Dr. José Lucas de Souza for the description of the ocular lesions, Dr. Daniel Lopes who performed the examination of the nose, mouth, and throat, and Mr. A. Esteves for the drawing of the ocular lesions found in my patient.

References

Brocq, L.: Pemphigus. Pratique Dermatol., **3**:770, 1902.

Church, R. N., and Snedden, I. B.: Ocular pemphigus. Brit. J. Dermat., **65**:235, 1953.

Gougerot, H., Blum, P., Carteaud, A., and Meyer, J. J.: Epidermolyse ou pemphigus traumatique d'apparition tardive. Bull. Soc. franç. de dermat. & Syph., **57**:501, 1950.

Lever, W. F.: Pemphigus conjunctivae with scarring of the skin. Arch. Dermat. & Syph., **46**:875, 1942.

Lindemberg, A.: Etiologia do fogo selvagem. Arch. Hyg. & Saude Publ. São Paulo, **2**:633, 1937.

Marie, J.: Le comportement des maladies entre elles. Presse mèd., **61**:187, 1953.

Melczer, N. Sulla eziologia del penfigo cronico e della dermatite erpetiforme. Arch. Ital. Dermat., Sif. & Venereol., **26**:32, 1954.

Rabelo, F. E., Portugal, H., and Azulay, R. O.: As buloses do grupo do pênfigo: suas correlações clinicas, funcionais e histológicas. An. V. Reunião dos Dérmato-sifilógrafos Brasileiros, Imprensa Oficial da Bahia, 1949, p. 132.

Radaeli, F.: Penfigo e penfigoidi. Giorn. Ital. Dermat. & Sif., **1**:60, 1932.

Ramel, E.: Sur une dermatose voisine du pemphigus à évolution cicatricielle essentielle. Ann. Dermat. & Syph., **7**:403, 1924.

ELECTRIC-ARC WELDING AMBLYOPIA*

Report of a Case

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and

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Of the different types of amblyopias, toxic, hysterical, and so forth, electric-arc welding amblyopia is now, practically, nonexistent in the literature. This is perhaps due to effective strict protective measures among welders in large factories so that it has not become a problem to industrial ophthalmologists. However, in smaller shops where the use of protective goggles is at times disregarded due to laziness or ignorance, the hazard appears every now and then.

Interviewing some welders in our locality, it was found that short exposures without goggles brings about transitory dimness of vision which lasts for a day or so and becomes a common-place experience which does not disturb them.

In a personal communication Dr. Hedwig S. Kuhn wrote:

> I am very much intrigued with your letter of February 21, 1957, and I wonder if perhaps the fumes of welding might have had something to do with the eye picture, as ultraviolet rays themselves do not penetrate the cornea and cannot cause any intraocular pathology themselves. Some of the new metals used in both electric and oxyacetylene welding have exceedingly toxic fumes which are inhaled and do have systemic effects. I certainly would make every effort to find out what the material was that was used in the electrodes either by the individual himself or people near him.
>
> If the ultraviolet rays themselves had produced any ocular damage at all, it would have been a corneal burn of considerable degree, which would in 24 hours or so have sloughed off the epithelial layer, just as a sunburn does, but I am sure that none of the findings you described have anything to do with ultraviolet radiation.
>
> While I am exploring the various metals that might have been in use, and getting some information along that line, I would appreciate it if you would also do so and let me know by return mail, other things that have to deal with the work environment and the individual. There is no known case of intraocular damage such as you have described, even experimentally that I know of and I have really searched the literature on a number of occasions. It might be well, if it has not already been done, to get a blood picture and any signs of chest or other findings that might do with a toxic condition emanating from the metallic fumes. This, of course, is a distinct possibility. This is a most unusual picture.

Case Report

A 20-year-old young man, emotionally unstable, an engineering student, who should have known better than to hold a piece of machinery being welded for about an hour without protecting goggles, came to my office the next day with bilateral vision of light perception only, no projection. The external eye appeared essentially normal except for slight conjunctival injection, the corneas clear, the pupils moderately dilated with very sluggish re-

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* From the Eduarda Bautista Research for Eye, Ear, Nose, and Throat. Abbott Laboratories, Chicago, Illinois, kindly donated the histamine diphosphate.

† Resident at Chong Hua Hospital.

action to light. The lens and vitreous were clear. There was moderate retinal edema. The most prominent picture was marked arteriolar vasospasm. Tension (Schiøtz): O.U., 17 mm. Hg.

The same day he was hospitalized and given the following: Cyanocobalamin (vitamin B_{12}) 1,000 mg. per cc., intravenously; Cortone, 100 mg., intramuscularly; prednisone (Meticorten) 5.0 mg., three times daily; vitamin E tablets (Ephynal) 100 mg. three times daily; Priscol tablets, three times daily; and nicotinic acid tablets, 50 mg. three times daily. After five days of this regimen, vision improved only to hand movements at one foot. The fundus picture was the same.

In as much as the main visible pathologic process was marked vasoconstriction, on the sixth day all previous medications were suspended and histamine diphosphate, 2.75 mg. in 500 cc. saline, was administered at the rate of 20 to 30 drops a minute intravenously, depending on the amount of flushing of the face and the blood pressure.

The next day, the patient was able to count fingers at one foot. For the next four days, histamine diphosphate was continued, and each day the retinal edema became less and the arteriolar caliber larger. The patient was discharged from the hospital on the 11th day with vision of: O.U., 3/200, J12. Twenty-six days later, the vision improved to 20/100, J12; another 18 days, the vision became 20/40, J1. Repeated fields of vision showed progression from tubular constriction of both fields, to progressive enlargement, to almost normal. The fundus regained its normal appearance and no permanent primary or secondary damage could be seen in the cornea, the lens, and the macula during almost a year of follow-up examinations.

Comment

I am most grateful to Dr. Kuhn for her kind explanation of the pathology of this case. Toxic fumes inhaled might have caused the profound constriction of the retinal arterioles. Ultraviolet and infrared rays might have induced the vasospasm by reflex action secondary to irritation of the sensory nerves of the external eye. As there was no permanent damage to the cornea, the lens, and the macula, toxic fumes may have been the etiologic factor.

Summary

This is the report of a patient with electric-arc welding amblyopia who recovered after the use of histamine disphospate intravenously by the drip method.

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MANDIBULOFACIAL DYSOSTOSIS*

Or bilateral facial agenesia (Treacher-Collins syndrome)

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Mandibulofacial dysostosis or bilateral facial agenesia is a congenital deformity of the facial bones which has not often been described in the literature. Although the key manifestations are found in the eyes and ears, the syndrome is little discussed by ophthalomolgists or otologists.

According to Apt[1] a total of 121 cases were found in 69 articles up to 1956; out of 16 cases described in eight papers reported in the United States, only two reports were from American ophthalmologists. He also reported that he has seen four patients in less than a year at Wills Eye Hospital and encountered previously five more cases while at the Children's Hospital. This suggests that the disease is more common than one would surmise. Herewith is the report of a case encountered in 1957.

Case report

R. Y., a Filipino boy, six years of age, was admitted for the first time to V. Luna General Hospital, Armed Forces of the Philippines, on April 25, 1957. According to the mother, she noted abnormality in both ears since birth. As the child grew older, she observed deafness in both ears for which she sought admission for her boy. Family history and past illnesses are irrelevant to the present complaints.

Physical examination of the child revealed him to be fairly well developed, fairly nourished, afebrile, and ambulatory. He could not speak but only made sounds. He understood signs very well. He was intelligent, active, and jolly. Other parts and organs of the body not described were found to be normal.

Eyes. The appearance and shape of the palpebral fissures appeared irregular, due to the presence of the following conditions:

1. In both upper lids, almost at the center of the left lid and a little medial of the center on the right,

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* From the V. Luna General Hospital, Armed Forces of the Philippines.

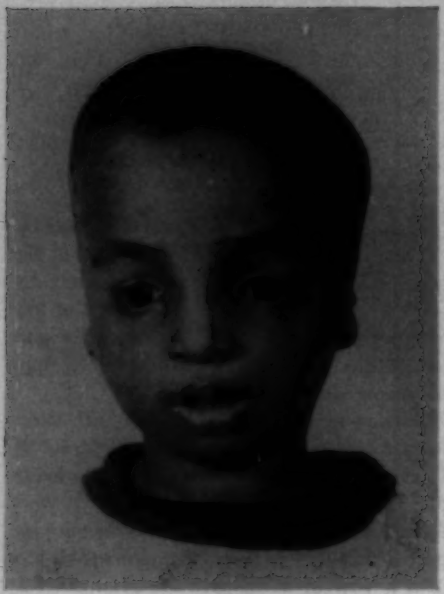

Fig. 1 (Sayoc). Anterior view.

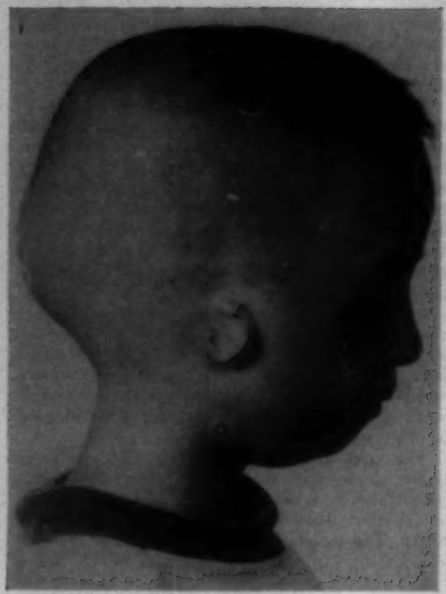

Fig. 2 (Sayoc). Right lateral view.

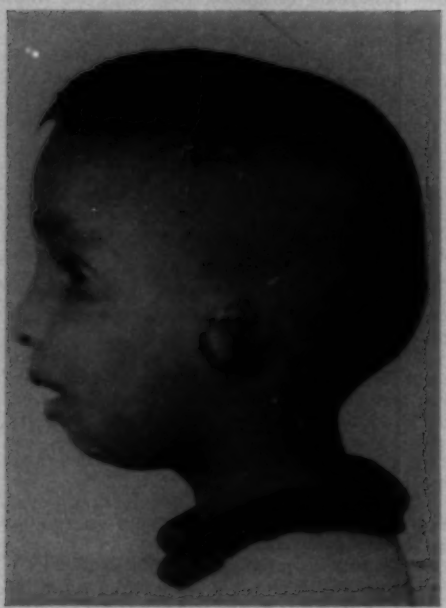

Fig. 3 (Sayoc). Left lateral view.

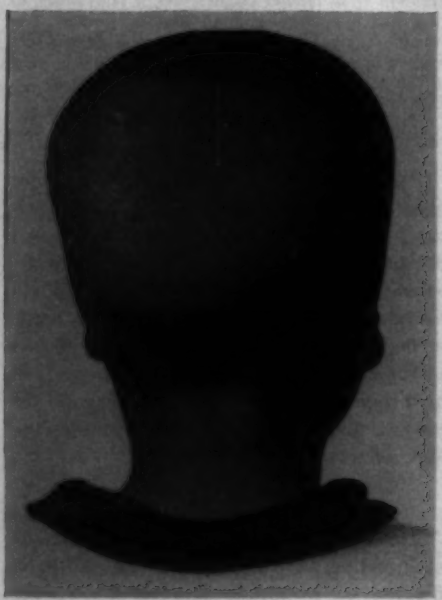

Fig. 4 (Sayoc). Posterior view.

were colobomatous deformities which gave the upper lids the appearance of irregular edges without eyelashes at the depressions.

2. Both lower lids had notching and colobomatous deformities accompanied by mild ectropion, producing a somewhat oblique palpebral aperture. The lower lids appeared thin and atrophic, with the medial half presenting abnormal lid margins and absent eyelashes. The punctas, however, appeared normal.

3. The vision was binocularly good, motility normal, and appearance of the eyeballs normal (fig. 1).

Ears. There was partial agenesia of both external ears. On both sides, a rudimentary portion of the helix was attached to the lobules which were themselves deformed. Both external meati and canals were absent, with deafness (figs. 2 and 3).

Nose. Externally and internally, the nose was normal. Respiratory passage was normal. At first glance, the nose appeared a little oversized for the face, due to underdeveloped maxillas and absence of the malar prominences (fig. 1).

Teeth. There was irregularity of tooth spacings (fig. 1).

Maxilla and malar bones. As can be seen in Figure 1, there was a hypoplastic maxilla with noticeable depression in the malar areas of the face. X-ray films revealed depressed malar bones and small antra.

Mandibles. There was micrognathia with manifest asymmetry. As can be seen in Figures 1, 2 and 3, the receding chin was most apparent.

Occipital bone. The external occipital proturberance was visible and palpable (figs. 2, 3, and 4).

This case was referred to Maj. Juanito Duque, chief of plastic surgery of this hospital, for whatever plastic and reconstructive surgery[3] he might elect. After making a thorough study of the case, the child was discharged with plans to reconstruct his external canals and auditory canals in the near future.[3]

Comment

Although this case did not present all the deformities that are included in the syndrome, the majority of the most important and classical features of Treacher-Collins syndrome are present which, when grouped together, substantially contribute toward a diagnosis. After having diagnosed his first case, a physician will not fail to recognize another case of bilateral facial agenesia.

V. Luna General Hospital.

References

1. Apt, L.: Arch. Ophth. **58**:288 (Aug.) 1957.
2. Snyder, Clifford: Am. J. Surg., **92**:81-87 (July) 1956.
3. Snyder, C. C.: Reconstructive surgery in children. Florida State J. Med., **42**:635, 1956.

CONTAMINATION FROM EYELASHES AND EYEBROWS

In intraocular surgery

F. H. Newton, M.D.
and
R. Grady Bruce, M.D.
Dallas, Texas

Several methods of preventing contamination of gloves, sutures, and instruments by contact with lashes and brow have been used. Cutting of the lashes and shaving the brow are common practices. It must be remembered that the bases of the lashes harbor many organisms capable of producing infections and cutting the lashes really gives the suture material a better chance to contact this area. Various specula have been designed to cover the lashes. For years we have been covering the brow with a piece of flat dry gauze sewed to the skin just below the brow line and turned up underneath the eye sheet. This very satisfactorily takes care of the hairs in the brow without any shaving.

For a long time the general surgeon has been protecting the operative wound from the surrounding skin by clamping towels to margins of wound and turning towels back over clamps. It was the suggestion of one of us (G. B.) that we apply this method in covering the lashes of both upper and lower lids. Lately we have followed this plan.

The lids and globe are immobilized by the operator's favorite method of anesthesia. We combine the O'Brien and Van Lint methods for the lids and retrobulbar injection for the

globe, waiting 10 to 15 minutes for the full effect of anesthetic.

With an eye sheet in place a silk traction suture is introduced in the center of the upper lid with a horizontal bite eight mm. long located four mm. above the lid margin and including the skin, subcutaneous tissue, and, possibly, the orbicularis. Then the edge of a square of moist gauze, well squeezed out, is attached snugly to the margin of each lid just posterior to the lash line with interrupted 4-0 silk sutures so placed that the knots are covered when the flap of gauze for the upper lid is turned up over the traction suture and eye sheet and that of the lower lid is turned down. The upper flap also takes care of the brow. This gives a good exposure with no pressure on the globe and with the lashes of both lids entirely covered.

When the superior rectus suture is later inserted it is drawn up over the gauze flap; thus, this suture and the traction suture of the upper lid are separated and easily differentiated. A traction suture in the lower lid is not necessary since the gauze flap can be used for traction if needed.

On completion of the operation the gauze flaps are removed and the operated eye is closed. As yet there has been little or no reaction on the lids and no interference with the meibomian glands.

We do not propose this as an entirely original procedure for in the past rubber tissue has been used in a similar manner. However, it seems worth while to bring this simple technique to the attention of ophthalmic surgeons since it is not in general use.

209 Medical Arts Building (1).

A DISCUSSION OF THE MECHANISMS IN CHRONIC ANGLE-CLOSURE GLAUCOMA

JULIUS KESSLER, M.D.
New York

The mechanisms active in primary paroxysmal angle-closure glaucoma have been outlined in a previous article.[1] In addition to a shallow anterior chamber and the increased thickness of the iris in pupillary dilatation, an increase of peripheral protrusion of the iris with pupillary dilitation, due to weakness of this part of the iris, has been suggested as an angle-closing factor in some of these eyes. This condition is manifested in eyes without marked shallowness of the anterior chamber and, nevertheless, prone to attacks of angle closure. A subaverage resistance of the peripheral iris is of no importance in eyes with a deeper anterior chamber but it may contribute to closure of the angle in eyes with more shallow anterior chambers.

In the course of primary angle-closure glaucoma the depth of the anterior chamber decreases progressively, due to the physiologic and often, also, to some pathologic increase of the size of the lens, as well as to some slackening of the zonule with increase of the size of the lens.

The thickness of the iris decreases with progressing age and prolonged disease and so does the resistance of the iris. Therefore, in a later stage of primary angle-closure glaucoma the contribution to closure of the angle by increase of the peripheral protrusion of the iris may be more important than in earlier stages.

In eyes with angle-closure glaucoma of long standing and under the influence of miotics, the whole iris is protruded, especially its periphery. In some eyes this protrusion is irregular showing a more localized weakness of some parts of the peripheral iris. The iris is tense and its surface is smooth. Optical section with the slitlamp through an iris of this kind and with little pigment often shows a very thin periphery.

The periphery of the iris of old persons is often thin and stretching by miotics makes it thinner. Atrophy by the disease itself and by continuous stretching due to the use of miotics may contribute to the thinness. Early stages usually show average thickness of the iris. The lower resistance of the iris mani-

fested by a peripheral protrusion may be due to a subaverage strength of the muscular or vascular layers.

Attacks of angle closure may stop when the mechanism which produces the closure of the angle is no longer fully effective. This can be due to structural change of the angle, to change in the closing force, or to prevention of attack-precipitating influences. The acute attack is due to a circular closure at the entrance of the angle which results in a complete closure of the ways of outflow in the angle of the anterior chamber.

Changes of the iris and of its anterior surface in the course of the disease may prevent a tight circular closure of the entrance. But loss of resistance of the root of the iris—this resistance helps to keep the space of the angle open[2]—may cause some attachment of the root of the iris to the trabecula. Anterior synechias, which may exist as the result of former attacks, may produce permanent partial closure of the angle of the anterior chamber. The area of attachment may increase with pupillary dilatation, and may decrease with pupillary constriction.[1]

The force which closes the angle is the difference of pressure acting on the anterior and posterior surfaces of the iris. It is due to the movement of fluid from the ciliary body to the angle of the anterior chamber and to the contraction of the pupillary sphincter muscle.

Partial obstruction of the ways of outflow does not decrease the movement of fluid. A higher intraocular pressure assures the same rate of flow. With reduction of the formation of fluid in later stages, due to atrophy of the ciliary body, the force acting on the iris decreases, and the tendency to closure may diminish. Reduction of the rate of flow reduces the protrusion of the iris and the tendency to closure of the angle.

Drugs reducing the rate of flow like Diamox, may be contraindicated in angle-closure glaucoma (because of undue delay of necessary and effective surgery) but these drugs do not affect the course of the disease in an adverse way.

Decrease of the force of the pupillary sphincter reduces the force acting on the iris but promotes closure of the angle by decreased stretching of the iris. Acute attacks may be permanently ended in some of these eyes by use of miotics at appropriate intervals. Miotics increase the force acting on the iris and may in the long run promote a distention of the iris and a shallowing of the anterior chamber.[2] Nevertheless, the eye may be controlled for years, with normal intraocular pressure and without attacks, although the iris is markedly protruded and atrophic and the angle very narrow but still free for outflow.

Some of these eyes, after prolonged use of miotics, show a partial obstruction of outflow due to some attachment of a weak root of the iris to the trabecula. In these eyes miotics may produce a quiet and delayed transition from the acute to the chronic stage of angle-closure glaucoma.

In some eyes with a configuration of the chamber angle which predisposes to closure, attacks never occur. This may be due to structural peculiarities of the peripheral iris which affect its resistance and its anterior surface. In some cases, use of miotics in both eyes after a glaucomatous attack in one eye may prevent an attack in the other eye. Some of these eyes, although a complete closure at the entrance of the angle does not occur, may experience a partial obstruction to outflow due to attachment of a part of the peripheral iris or only of a part of the root of the iris to the trabecula. The area of attachment may increase with pupillary dilatation. There is a weakness of a part of the peripheral iris and of the root of the iris in these eyes.

In the type of angle-closure glaucoma in which attacks recur, the closure starts at the entrance ring of the angle. Minimum patency of this ring permits access of fluid to the whole area of the trabecula. Subminimal patency or closure means the difference between free outflow and complete obstruction. The resistance of the root of the iris is sufficient to keep the space of the angle open (unless this space is completely separated from

the anterior chamber proper) and to open the space when the communication is restored. In such cases, complete angle-closure is intermittent.

In the chronic type of angle-closure glaucoma the entrance ring of the angle may be partly closed or entirely open. The obstruction to outflow is not due to closure at the entrance but to more or less attachment of the root of the iris to the trabecula, with more or less obstruction to outflow, corresponding to the area of attachment. The attachment may be by synechias or by apposition of a weak root of the iris. The resistance of the root of the iris or of a part of it is not sufficient to keep the space of the angle open, even when this space is in communication with the space of the anterior chamber proper. There is a permanent incomplete angle closure.

229 East 79th Street (21).

REFERENCES

1. Kessler, J.: Mechanisms in angle-closure glaucoma. Am. J. Ophth., **43**:271, 1957.
2. ———: The resistance to deformation of the tissue of the peripheral iris and the space of the angle of the anterior chamber. Am. J. Ophth., **42**:733, 1956.

REOPENING TECHNIQUE FOR CICATRIZED TREPHINATION

HAROLD F. WHALMAN, M.D.
Los Angeles, California

In times past I have made attempts to reopen the cicatrized trephined area by means of blunt instruments, such as a cyclodialysis spatula, only to encounter such resistance as to make it impossible to reach the opening, with more trauma and more cicatrix being produced. A suitable sharp instrument was sought but small cataract knives, Wheeler knives, and even needle-knives conformed so poorly to the curvature of the eyeball that the trephined area could not be satisfactorily reached through a small puncture wound in the conjunctiva.

Recently a goniotomy knife has been used with success. The conjunctiva is injected with Novocaine at the 12-o'clock position and a tangential puncture made one cm. temporally. When the blade reaches the trephined area, it is held snug to the sclera and a few strokes readily cut the cicatrix free. Air may then be injected beneath the conjunctiva until a bubble is observed in the anterior chamber. The air injection was particularly serviceable in pushing back the vitreous in an aphakic eye which had been trephined for glaucoma and later had a successful intracapsular cataract extraction through an incision at the 6-o'clock position.

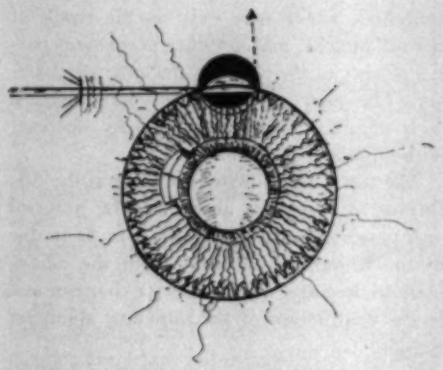

Fig. 1 (Whalman). Drawing, showing the goniotomy knife inserted beneath the conjunctiva and over the trephining area.

727 West Seventh Street (17).

OPHTHALMIC RESEARCH

EDITED BY FRANK W. NEWELL, M.D.

Abstracts of papers presented at the meeting of the Southern Section of the Association for Research in Ophthalmology, Inc., at the Roosevelt Hotel, New Orleans, Louisiana, November 3, 1958

A. E. Meisenbach, Jr., M.D., Dallas, Texas
Section Secretary

Irritation studies of methylcellulose. T. C. Fleming, M.A., and D. L. Merrill, M.S., Alcon Laboratories, Inc., Fort Worth, Texas, and Louis J. Girard, M.D., Department of Ophthalmology, Baylor University College of Medicine, Houston, Texas.

Methylcellulose to increase viscosity has been included in many ophthalmic preparations since 1945. Using laboratory animals, subcutaneous and intraperitoneal injections gave no indications of irritation or sensitization. Intraocular injections of 0.1 ml. of a 0.5-percent solution of methylcellulose in a balanced salt solution gave no indication of irritation. Qualitative tests of the aqueous after injections of methylcellulose into the anterior chamber gave negative results for methylcellulose after the fourth day. Tissue culture studies using human conjunctiva cells in a 0.5-percent concentration of methylcellulose resulted in normal cellular growth. Microscopic examination of tissues from the anterior chamber which were subjected to methylcellulose over a period of 10 days gave no indication of a tissue reaction to this material.

Therapeutic studies in experimental chemical injury of the cornea: II. Phosphate buffer (Neutralize) studies. Gustav C. Bahn, M.D., Earl Sonnier, M.D., and James H. Allen, M.D., Tulane University, New Orleans.

Management of chemical injury of the cornea falls into two phases: (1) emergency care or first aid, which is designed to reduce the severity of the burn by prompt removal, neutralization, or counteraction of the noxious agent, and (2) by definitive medical or surgical means, to promote healing without complication and/or replacement of damaged or scar tissue with that which is functionally more normal.

The purpose of this report is to evaluate the results of a series of experiments surveying the effectiveness of a phosphate buffer system used as an emergency means of treating representative acid and base burns of the cornea.

The corneas of albino rats were burned, employing the method previously described by Bahn and Allen. A modification of the Friedenwald scheme of evaluating the course of the burns and efficacy of treatment was followed. All animals were kept under observation until the healing process became stationary.

Two forms of the phosphate buffer solution were used. The first contained 0.5-percent chlorobutanol as a preservative. Because of the reported inhibitory effect of chlorobutanol upon epithelial regeneration, a group of experiments were conducted using the buffer solution without preservative, the solution being freshly autoclaved before use, to insure sterility.

The phosphate buffer (Neutralize), when used in small amounts after acid and alkali burns of the cornea, decreased the severity and increased the healing rate of the burn more than did an equal amount of distilled water. It was noted that small amounts of Neutralize were as effective as larger amounts of distilled water, by a factor of about 1:15. Thus, Neutralize, if quickly applied, will to some extent protect the cornea from acid and alkali burns, although the same degree of protection can be obtained by profuse distilled water irrigation. In no case were any detrimental effects produced by Neutralize. No difference was noted between the effect of Neutralize with and without chlorobutanol added as a preservative. These experiments tend to indicate the clinical usefulness for the phosphate buffer solution, Neutralize, especially as recommended, in the field or under conditions where water for irrigation purpose is unavailable. No superiority to adequate irrigation with distilled water was demonstrated.

Evaluation of dichlorphenamide as an ocular hypotensive agent. Robert A. Schimek, M.D., Ochsner Clinic and Tulane University, New Orleans, Louisiana, J. V. Balian, M.D., F. J. Lepley, M.D., and J. A. Ottum, M.D., Department of Ophthalmology, Henry Ford Hospital, Detroit, Michigan.

The value of the carbonic anhydrase inhibitor, acetazolamide, in lowering intraocular pressure has already been proved. It follows that the newer carbonic anhydrase inhibitors might also have similar ocular pharmacologic action. Dichlorphenamide and chlorothiazide (Merck, Sharp, and Dohme) were chosen for experimental investigation in this study.

1. In the preliminary phase of the experiment, controls were not used. Tonometric measurements were made on New Zealand white rabbits for several days before drug administration and at three hours and six hours after administration of dichlorphenamide and chlorothiazide. These pre-

liminary results appeared to indicate a lowering of intraocular pressure but later, more carefully controlled experimental studies indicated that this result was probably biased. Subsequently, with "controlled" experiments, no hypotensive effect of the drugs could be elicited unless there was previous administration of steroids to cause the rabbits to become "responsive."

2. A second experiment, utilizing the Roman-square method of statistical investigation, was set up with controls, and the investigators performing tonometry had no knowledge of which rabbits were controls and which were treated. Ten rabbits in two Roman-square designs served alternately as controls, or received dichlorphenamide, chlorothiazide, or acetazolamide. Each rabbit was alternately treated by the various drugs or served as a control through five separate "runs." Statistical analysis showed no significant effect on intraocular pressure from Diamox, dichlorphenamide, or chlorothiazide.

3. Experiment 2 was repeated with 15 rabbits, with no significant effect on intraocular pressure from any of the drugs as shown by statistical analysis.

4. Becker has shown that adrenal steroids prevent compensatory decrease in outflow facility of rabbits, which may occur following acetazolamide administration. To assure "responsive" rabbits, 9-alpha-fluorohydrocortisone was given to 10 rabbits which were studied in four consecutive trial periods. Each rabbit alternated between receiving dichlorphenamide and not receiving it in the next period. The investigator taking the tensions was unaware of which rabbit received the drug. Statistical analysis showed that the administration of 9-alpha-fluorohydrocortisone itself had no significant effect upon the intraocular pressure. However, the data did indicate a significant (PN, 0.00001) lowering of intraocular pressure one hour following parenteral administration of dichlorphenamide, when the rabbits were made "responsive" by steroid administration.

5. A group of eight rabbits treated with dichlorphenamide showed a significant decrease in the bicarbonate ion concentrations of the posterior chamber aqueous even when they were not given 9-alpha-fluorohydrocortisone.

The importance of carefully controlled experimental studies was demonstrated in this evaluation of the hypotensive effect of drugs as determined by rabbit tonometry. New Zealand white rabbits in our experiment, on a normal rabbit diet and without special medication, did not demonstrate a significant lowering of intraocular pressure after oral acetazolamide administration. This is in agreement with reports that certain rabbits are "pseudoresistant" to the hypotensive ocular effects of the drug. These rabbits were also treated with dichlorphenamide and did not show lowering of intraocular pressure on a normal diet without supplementary medication. Thus, certain groups of rabbits may be "pseudoresistant" to any carbonic anhydrase inhibitor alone. Administration of 9-alpha-fluorohydrocortisone caused our rabbits to become "responsive" to parenteral dichlorphenamide and show a significant decrease in the intraocular pressure. Dichlorphenamide was also found to cause a significant decrease in the bicarbonate ion concentration of the posterior chamber aqueous of rabbits even when they were not given 9-alpha-fluorohydrocortisone.

Postoperative keratitis following cataract extraction. Louis A. Brefeilh, M.D., Division of Post-Graduate School of Medicine of Louisiana State University Medical School at Confederate Memorial Hospital, Shreveport, Louisiana.

This paper is concerned with the problem of postoperative keratitis which frequently follows cataract surgery. This condition may be only slight in the region of the incision and remain as a minor annoyance to the patient's comfort or an impediment to vision. Often the condition becomes bullous due to the corneal edema and results in corneal opacification with visual loss. In either event the patient is dissatisfied with the results. This condition of bullous keratitis occurred in two successive cataract patients and resulted in my investigation of this problem.

All available cataract patients were restudied. Cataracts performed by other surgeons were studied. The corneas of all patients who were to undergo surgery were studied. Finally, corneal incisions were made at various sites during the cataract extraction operations. The slitlamp studies of corneas of preoperative patients revealed numerous endothelial and epithelial changes, which I felt could result in keratic edema. Fortunately, none of these patients developed the postoperative keratitis. The results of this phase of the investigation indicated that the condition was not necessarily a result of corneal degenerative conditions.

Cataracts performed by me revealed that two in 204 cataract extractions developed bullous keratitis and were failures. The cataract patients operated upon by other surgeons were then studied. Six had failures due to bullous keratitis and three had subnormal vision due to keratitis near the incision site.

The clinical investigation disclosed that the site and subsequent closure of the wound are important factors. The next series of cataracts that were performed were classified as to the site of the incisions. The patients who had their incisions in the scleral side of the limbus developed only transient edema and their corneas remained clear after healing.

My investigations indicated that the best site for the incision is one mm. posterior to the limbus. The wound closure seems to be aided by the vascularity in this area and the postoperative corneal edema is transient and visual improvement is more rapid.

Operations in seven patients were failures be-

cause of bullous keratitis. One was my patient and six were referrals. All these patients obtained clear postoperative corneas in the second eyes. The visual results were 20/25 or better except in a 93-year-old woman with marked retinal arteriosclerosis. By using this site for the incision, I have not had a cataract failure due to surgical technique in the last three years.

Inheritance of congenital anophthalmia in mice. Aeleta Nichols Barber, Ph.D., Louisiana State University School of Medicine, New Orleans, Louisiana.

Investigations in experimental teratology are usually conducted on animals having a normal or unknown genetic background and the resultant abnormalities are attributed entirely to the toxic effects of the treatment. Very little attention has been given to the effects of teratogenic agents on the inheritance of a known genetic complex. The animals used in the experiments to be reported in this paper have a genetic background of bilateral anophthalmia. When these mice are mated with normal mice, the first generation hybrids always have normal eyes even though they carry the recessive gene for anophthalmia. Treatment with trypan blue, cortisone, and antigens during gestation affects the expression of the genetic complex in different ways.

Changes in bacterial drug sensitivities. Thomas S. Edwards, M.D., Jacksonville, Florida.

In this project approximately 2,000 bacterial cultures were taken from the eye, nose, and throat over the three-year period. The cultures taken were evenly divided between hospital patients and private office patients. The drug sensitivity of these cultures was tested against six of the more common antibiotics and the results tabulated. The differences between the two sources, the antibiotics, and the various time intervals were discussed.

Ophthalmic Miniature

I introduce a broad needle through the cornea, close to its junction with the sclerotic, and allow the aqueous humor to escape. I then draw out a portion of the iris with a blunt hook, and leave it in the wound; or if it protrude much, I remove a portion and leave the remainder in the wound. The immediate effect of this operation is to remove the tension of the globe, and relieve the pain; the secondary effect is the gradual and steady improvement of the sight; and as far as I can at present judge, the remote effect is the removal of the tendency to fresh attacks.

Mr. Critchett on "Treatment of acute glaucoma,"
Ophth. Hosp. Reports, 1:58, 1858.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

CHICAGO OPHTHALMOLOGICAL SOCIETY

April 21, 1958

FRANK W. NEWELL, M.D., *President*

CONGENITAL GLAUCOMA

DR. A. EDWARD MAUMENEE reviewed the currently accepted theories of congenital glaucoma. These are:

1. An absent canal of Schlemm; in almost all sections of eyes with congenital glaucoma a more or less patent canal of Schlemm is visible.

2. Persistent mesodermal tissue; Dr. Maumenee felt that this is often erroneously seen gonioscopically since it is almost impossible to demonstrate this tissue in histologic preparations. Furthermore he quoted the work of Burian, et al., who showed that there was no decrease in the number of cells in the angle in the adult eye as compared to the embryonic eye.

3. Anterior insertion of the iris; Dr. Maumenee felt that this accounts for about five percent of the cases of congenital glaucoma.

4. Choroidal inflammation; this has not been shown to be a factor.

Having eliminated these, Dr. Maumenee presented his own theory based on the histologic examination of over 40 eyes with congenital glaucoma. He demonstrated that in many cases the meridional fibers of the ciliary body insert into the trabecula in front of the scleral spur. On contraction, these anteriorly inserted fibers are capable of collapsing the canal of Schlemm and thus blocking the outflow of aqueous.

Discussion. DR. JOSEPH HAAS: I am very grateful to Dr. Maumenee, for it was he who first aroused any interest that I may have in infantile glaucoma and I have since followed the course of the various schools of thought regarding infantile glaucoma. Dr. Maumenee has shown Dr. Barkan's original pictures depicting a rather heavy mesodermal obstruction to outflow, and then his later modification, in which he speaks of the cellophanelike membrane covering the trabecula, preventing the outflow. He has shown some of his very magnificent angle pictures in which originally he believed the anterior insertion of the iris root might have been a factor in the outflow. Now, he has come up with an original and exciting theory, that it is the anterior insertion of the muscles of the ciliary body which prevents the opening of the trabecula and thus prevents the aqueous from reaching the canal of Schlemm.

I certainly think that this explains the situation better than any theory that has been advanced to date. I am in agreement with Dr. Maumenee that the gonioscopic evaluation of these angles is extremely difficult. I think that there are certain questions that remain to be answered completely but I shall not mention them tonight because this theory is certainly the greatest approach to answering these questions that has been made.

I think, gonioscopically, the angle picture differs considerably and, therefore, I would agree with Dr. Maumenee that there are probably several etiologic factors which may be responsible for the production of infantile glaucoma, ranging from the classical obstruction by the anterior insertion of the iris that you see in Axenfeld's syndrome to the gonioscopic picture with no obstruction whatsoever.

I don't really know if the anterior insertion of the ciliary body can obstruct these trabecular spaces but it is the most exciting thing that I have heard regarding the pathogenesis of the condition and the explanation of the goniotomy operation. It is my impres-

sion that occasionally in some of these cases, without too high a tension, it is possible to fill the canal of Schlemm preoperatively before normalization of the pressure and, if this be so, then it could be said that it is only a partial pull or a partial resistance of the trabecular spaces that prevents the absorption of aqueous, which would fit with this theory very well.

Another thing that puzzles me somewhat is why these cases seem always much more severe when the disease occurs early and also is much more severe when the globes are already enlarged. That probably could be explained by the fact that the muscle would be farther forward in those cases.

I would like to compliment Dr. Maumenee. I know of his great interest in this condition, and I think that this report is certainly the most exciting thing that has happened in the field of infantile glaucoma.

DR. C. RUDOLPH: There are a few things that bother me. The first is that Dr. Maumenee seemed to throw all congenital glaucomas into the same category. I have always been taught to differentiate between a hydrophthalmos and a buphthalmos. They are certainly pictured grossly as quite different. The second thing is whether these muscle fibers anterior to the scleral spur are a result or the cause of glaucoma. If it was due to an intrauterine inflammation, one would probably find inflammatory cells and exudate present but, according to the microscopic section demonstrated, there were none.

DR. A. EDWARD MAUMENEE: I would like very much to thank the discussers for their comments. As so frequently happens when one considers a microscopic field, one can spend hours and days discussing it and not just 30 minutes. There are so many, many other points that could be brought out on this subject that I just didn't have time to go into very thoroughly.

First of all, in order to clear up what I should have mentioned in my opening sentence, Dr. Rudolph, I agree with you completely, that there are very many types of glaucoma. What I was speaking of is the so-called primary glaucoma that we don't have any obvious explanation for—such as uterine uveitis. Furthermore, I feel (and this is not original with me. I think it goes back to Treacher-Collins) we should get rid of the terms buphthalmos and hydrophthalmos. Those are poor terms. We should substitute primary and secondary congenital glaucoma, the primary being those cases that we don't have any obvious explanation for, such as inflammation.

Then to come to some of Dr. Haas' points. Necessarily, I think anyone who tries to interpret from morphologic pathology is sticking his neck out but the only real fun of doing pathology is to stick your neck out, so, therefore, I wanted to put some explanations of physiology into these morphologic changes. I think beyond a doubt, we are going to have to prove or disprove this through physiologic experiments and not purely through microscopic studies for, once an eye is dead, one can't show how much that muscle pulls. There is some evidence, however, that this may be clinical—some physiologic evidence. One bit of evidence is that, in congenital glaucoma, there are exacerbations and remissions of the disease.

Recently there was a worker in Canada who showed that, when these children go to sleep, they have an amazing fall in intraocular pressure—showing that, when the muscle relaxes, possibly the canal and trabecular fibers open up. There has also been reported the interesting finding that giving atropine to children with early congenital glaucoma has controlled the tension when pilocarpine and eserine wouldn't. And all of us know how poorly these children are controlled by pilocarpine and eserine for any appreciable period of time.

As to the question of severity of the disease in early cases and in those with enlargement of the globe, one can only suppose that here the trabecular meshwork is completely and continuously closed rather than partially and intermittently.

PARESIS OF THE INFERIOR RECTUS MUSCLE

DR. BEULAH CUSHMAN AND DR. WARREN BINION reported 62 cases of inferior rectus paresis. Eight of these had only a vertical anomaly while the remainder were about equally divided between associated esotropia and exotropia. Occlusion is often helpful in eliciting an obscure vertical anomaly. Treatment is a resection of the paretic inferior rectus. Of the 62 cases, seven required a second procedure and two of these a third procedure. These cases were presented in detail and a movie shown to illustrate the preoperative and postoperative findings.

RETROLENTAL FIBROPLASIA

DR. CARL APPLE reviewed his experiences with premature infants during the past six years at Mount Sinai Hospital in Chicago. In this interval 252 prematures were examined and various types of oxygen therapy employed. Up to 1953 high oxygen concentrations were used. After this time little or no oxygen was employed. Dr. Apple concluded that the safest procedure is to use no oxygen therapeutically, if possible. In the first few days of life a concentration up to 60 percent may be used for a few hours but this should be quickly reduced. The most critical period for the development of retrolental fibroplasia is from the 27th to the 31st week of gestation.

David Shoch,
Recording Secretary.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

439th Meeting, December 18, 1957

VIRGIL G. CASTEN, M.D., *presiding*

IDIOPATHIC SEROUS DETACHMENT AND HEMORRHAGIC DISCIFORM DEGENERATION OF THE MACULA

DR. A. EDWARD MAUMENEE, Baltimore: The material on which this report is based is the personal records of 52 cases of idiopathic serous detachment and 100 cases of hemorrhagic disciform degeneration of the macula. Histologic examination has been made on 14 specimens of serous detachment and 60 specimens of hemorrhagic detachment of the macula.

The clinical manifestations, course of the disease, treatment of the condition, possible etiologies, and pathologic appearances of serous homorrhagic detachment of the macula were discussed.

Serous detachment of the macula is manifested by a localized elevation of the retina in an average area of about one and one-half to two disc diameters. The lesion is usually transient and subsides, with a resultant normal vision in 50 percent of the patients.

Prolonged detachment produces a cystic degeneration of the retina and permanent loss of vision.

The disease is usually unilateral and affects males four times more frequently than it does females. Examination of the clinical and histopathologic material indicates that angiospastic retinopathy is not a factor in this condition. Nor does a study of the eyes available for histologic examination confirm the impression that idiopathic serous detachment of the macula is caused by an inflammatory reaction.

Hemorrhagic disciform degeneration of the macula is a devastating condition as far as visual acuity is concerned. The lesion usually progresses to a permanent loss of vision at a level of 20/70 to 4/200. In at least 50 percent of the patients reported in this study the lesion was bilateral. Males are affected only slightly more frequently than females. The clinical manifestations vary a great deal from patient to patient and at different stages of the disease. Some bizarre forms of this lesion exist.

Hemorrhagic detachment of the macula may be confused with melanoma of the choroid and inflammatory chorioretinitis. However, in most instances these lesions can be differentiated if the complete picture of

hemorrhagic detachment is kept in mind.

The age distribution of idiopathic serous detachment of the macula and hemorrhagic detachment is approximately the same. Evidences of chorioretinal inflammation in areas distant from the macula were not found in serous detachment but were frequent in hemorrhagic detachment when the patients were under 45 years of age.

Histologic study of eyes that have been removed for hemorrhagic detachment of the macula does not reveal a significant degree of inflammatory cell infiltration in the choroid in most instances. The lesion is essentially due to a hemorrhage from the choroid into the subretinal area between the pigment epithelium and Bruch's membrane. No further clues are obtained as to the etiology of this lesion from the studies of the pathologic material.

It is thought that idiopathic serous detachment and hemorrhagic detachment of the macula should be grouped together in spite of the difference in their clinical course and the difference in the pathologic response. The reasons for grouping these two lesions together are (1) they both involve the macula; (2) they occur in patients of the same age group; (3) they appear to be due to some vascular disturbance; (4) in some instances a serous lesion is a prelude to a hemorrhagic detachment. Thus, a patient may have several attacks of serous detachment and then develop a hemorrhagic detachment of the macula. In other instances the two lesions may occur simultaneously but the primary response is a serous one which then progresses to a full-blown hemorrhagic detachment.

The etiology of these two lesions is not known at the present time. The various lesions with which both conditions are associated are (1) nongranulomatous iridocyclitis; (3) hypotony following cataract extraction; (3) macular detachment after cataract extraction; (4) melanomas of the choroid; (5) hypertensive retinopathy.

No satisfactory treatment for either condition is available at the present time. Further experimental and clinical studies of these diseases will have to be done before a rational form of therapy can be devised.

Comments. Dr. Charles L. Schepens: I must say that I am still unconvinced as to the clinical identity between central serous and disciform degeneration. Disciform is obviously a degenerative disease. What leaves me unconvinced is how can there be the central serous form and 20/20 vision? After repeated episodes, even after 10 years, there is still 20/20 vision. Another point is the frequency of the central serous form. Some members of our hospital staff have it and they have 20/20 vision. It is my impression that the central serous is different from disciform degeneration.

Then we must consider the edema of the posterior pole which may occur after cataract extraction and I think after any type of intraocular surgery. These lesions look entirely different from central serous or disciform degeneration because there is a more diffuse edema. It always involves the disc, usually first, and always leaves the vision impaired. With central serous degeneration the patients do retain good vision or normal central vision even after they have had the disease for some time.

Maybe there are two grades of the same disease but, clinically at least, or until more evidence is brought forward, I am not convinced on this particular point.

Dr. Robert Alpert: During the Berlin Air Lift I saw two or three very capable young aviators who had just been through a month of torture as far as work was concerned and these men had typical central serous retinopathy as it is described. Their response was good with rest. I just wonder if there could be some psychosomatic basis to all this.

Dr. Virgil G. Casten: When I was in the Pacific I saw a number of such cases. The patients all had cold, clammy hands, perspired easily, were excitable, and were of the neurasthenic type in many ways. They even had a normal retinal background and then this real dark, wine-red central opacity.

I have seen the same sort of thing since the war. What the findings were and what caused them I don't know.

Dr. Edwin B. Dunphy: I want to make just one comment: Always look at the other eye. A number of these cases are bilateral but not always bilateral at the same time. One fundus lesion may be well in advance of the other fundus lesion. I think this explains why a number of the eyes have been taken out mistakenly for malignant melanomas. The patient frequently makes no complaint of the other eye but close examination often may show the beginnings of the changes in the macula of that eye.

Dr. A. Edward Maumenee: First, about the changes in the two eyes. The longest time between the involvement of the first and second eyes in my cases has been 33 years and I have another case that has gone 32 years. So I think some patients can go a long time before the second eye is affected.

Dr. Schepens says he is not convinced that serous and hemorrhagic detachments are the same thing at all. I agree that most of the patients with serous detachment do not go on into the hemorrhagic or organized phase of the disease. Perhaps 80 percent get well within a year. A few go on to cystic degeneration and have poor vision. I want to point out that I have seen many patients, young and old, that had hemorrhagic disciform degeneration in one eye and typical serous detachment in the other eye. This condition in one eye and the other condition in the other eye makes one think that there must be some connection. The two conditions are at least first cousins even if they aren't brothers and sisters.

About postcataract patients: I have had a patient with mascular lesions in both eyes and both eyes with serous detachment. In the majority of cases we had, vision came back to normal.

There is a psychosomatic element in all tense people. Now during the war the civilians weren't affected by this eye condition that has been described. It seems to have been only the soldiers. I have wondered about this. Certainly both groups were under great strain.

Charles Snyder,
Recorder.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

Section on Ophthalmology

January 16, 1958

Harold G. Scheie, M.D., *Chairman*

Nervous control of intraocular pressure

Mr. E. S. Perkins, F.R.C.S., London, England: The necessity of some control of the intraocular pressure and the mechanisms whereby such control could be exercised strongly suggest that the nervous system is involved. By analogy with other pressure systems in the body it would be expected that the controlling nervous pathways would consist of pressure receptors in the eyes, a sensory pathway, a central area in the brain, and an efferent pathway.

A team of workers under the direction of Sir Stewart Duke-Elder has been investigating the evidence for such a controlling mechanism and, although it must be emphasized that to demonstrate that any particular nerve when blocked or stimulated causes changes in intraocular pressure does not prove that such a nerve is involved in the control of intraocular pressure, it has now been shown, at least in experimental animals, that nervous pathways do exist which could fulfill this function.

The sympathetic supply to the eye, the third, fifth, and seventh cranial nerves have all been studied, and the sympathetic and fifth cranial nerve have been shown to exert definite effects on the intraocular pressure. Stimulation of certain areas of the diencephalon may also result in changes in intraocular pressure, so that the efferent and central pathways probably exist. Little attention has been paid to the efferent or sensory pathways until recently, but it has now been

shown that changes in the intraocular pressure in cats do provoke potentials in the long ciliary nerves which can be amplified and recorded, suggesting that there may be pressure-sensitive receptors in the eye. If this finding can be substantiated there will be good evidence that the requisite pathways for a nervous control of intraocular pressure do exist, although the presence of such a control under normal conditions has not yet been proved.

William E. Krewson, 3rd,
Clerk.

Ophthalmic Miniature

What makes the cataract operation strenuous and delicate is the apprehension under which one labors and which one cannot suppress. I admit that I underwent one of the greatest humiliations of my life. The doctor had told me "Keep your eyes turned down and relax; don't get tense." What is easier than to follow this admonition? "This is easy," I had told myself, "I won't get tense since I have been told not to." And I held myself rigid with all my resources in order not to get tense. And as to the eye which should be turned downwards—it cannot be done! When I saw—and I still could see a little—when I saw the little devil of a knife move in front of my lids, my eye turned inevitably, incoercibly upwards.

"Look down," Dr. Perrin said softly.

"Look down," Dr. Félizet repeated, with a slightly shaking voice.

And I felt a contempt against myself, mixed with fury, for after having turned my eyes down with a terrible effort they slipped out of my control again and moved under the upper lid like shot from a gun.

"I am humiliated, doctor," I said, desperately nervous, "I am humiliated; I thought one was always master of one's movements. I am not. This is humiliating. What help is it to be a philosopher?"

"Be calm," Dr. Perrin said, "this is a reflex movement."

"I know all about reflexes, but I am not master of myself, I am humiliated!"

During this exchange Perrin had seized the eye, opened it and the lens escaped. The operation was done, and well done, with a rapidity and sure hand of which my friend, Dr. Félizet, still speaks with admiration.

"Can you see my hand?" the surgeon asked and moved it in front of my eye. I saw it in a kind of fog. "Yes, I can see it." "Well, that's it!" And he lifted once more the upper lid, looked carefully and, while he showed something to Dr. Félizet, he said "I don't like that!" Félizet was bent over me. I could not see either one, but I sensed that something had happened which might compromise the success of the operation. I felt like fainting. I know now that the iris had refused to contract and to assume its normal position. Perrin took a pair of scissors and with a firm movement cut, or rather trimmed, the prolapsed part of the iris. I felt a tremendous, lightening pain. My hand pressed that of Félizet, who had kept it firmly in his during the whole procedure. "All done, old man," he said, "it's done. You may rest quietly now."

Francisque Sarcey,
Gare á vos yeux!
Paris, 1884.

AMERICAN JOURNAL OF OPHTHALMOLOGY

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THE XVIIITH INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

The centennial celebration of the International Congress of Ophthalmology was held in Brussels, Belgium, from September 8 to 12, 1958, the city of its birth in 1857, under the high patronage of Her Majesty, Queen Elisabeth, and the General Commissariat of the Universal and International Exhibition. More than 2,000 ophthalmologists from practically every nation in the world, accompanied by wives, children, and guests, attended. The weather, though warm, was kind, the beautiful city was at its festive best, and the social events were brilliant and not to be forgotten.

On the evening of September 7th (Sunday), a reception of welcome in the beautiful museum, the "Cinquanteuaire," musées Royaux d'Art et d'Histoire, Avenue des Ner-

viens, Brussels, was held. An opportunity was afforded to meet here for the first time, the officers of the congress and their wives, some city fathers, and many friends from everywhere, promenading through numerous rooms of magnificent art treasures, mainly of the Middle Ages, and enjoying the bountiful refreshments that were provided.

At 10 o'clock the next morning, the official opening ceremony of the congress, under the presidency of Prof. Léon Coppez of Brussels, and in the presence of Her Majesty, Queen Elisabeth, took place in the Grande Salle de Concert, Palais des Beaux Arts. Professor Coppez gave the address of welcome, followed by that of Sir Stewart Duke-Elder who presented the Gonin Medal, in the name of the Queen, to our own Alan C. Woods of Baltimore, a most joyous event, particularly for those of us from the United States.

Then followed appropriate remarks by Léon Coppez; Sir Stewart, the president of the International Council of Ophthalmology; Dr. Maxwell-Lyons, representative of the World Health Organization; and the representative of the continents: for Europe, Prof. G. Renard (Paris); for North America, Prof. Derrick Vail (Chicago); for Asia, Prof. Naganori Kirisawa (Tokyo); for South America, Prof. Cyro de Rezende (São Paulo); for Africa, Dr. L. Staz (Johannesburg); and for Australia, Dr. J. Ringland Anderson (Melbourne).

In the afternoon, the scientific program opened with a symposium on radioactive isotopes in ophthalmology, under the gracious chairmanship of Prof. E. B. Dunphy of Boston, who discussed as the first paper, "The status of radioactive phosphorus in the detection of ocular tumors." Other opening speeches on the subject were Ullerich (Hamburg), Dollfus, et al. (Paris), Perkins, et al. (London), Palin (Bristol), and Appelmans and Wouters (Louvian). Ten free papers were given in discussion, three by ophthalmologists of the United States.

Concurrent with this, in another hall, 12 free papers on the subject, "Aqueous humor and retinal vessels," were presented under the joint chairmanship of Prof. V. Čavka (Belgrade) and Prof. N. Blatt (Bucharest). One United States member contributed. In still another hall, 12 free papers on "Diseases of the conjunctiva" were given (three from the United States). The chairmen were Prof. J. Verdaguer (Santiago de Chili) and Prof. Louis Sanchez Bulnes (Mexico).

That evening a scintillating reception and ball was held in the gorgeous medieval Hôtel de Ville. It was fairyland transplanted to the middle of the 20th century. It is rewarding to see what lovely women ophthalmologists seem universally to marry, and their beauty, and that of their gowns and jewels, formed an unforgettable picture.

The next morning (September 9th), a symposium on "The orthoptic treatment of concomitant strabismus," under the chairmanship of Prof. Alan C. Woods (Baltimore) was held. The opening speakers were A. Bangerter (St. Gallen), T. Keith Lyle (London), and J. Malbran (Buenos Aires). Thirty-five speakers with free papers took part in the discussion (four from the United States). Orthoptic treatment did not take the beating that I expected it to endure.

In the afternoon, the main topic for discussion was "The genesis of functional symptoms in an eye with raised tension," chairman, Sir Stewart Duke-Elder. There were seven opening speakers (none from the United States) and 16 free papers (two from the United States). In the meantime, in another hall, the subject of "Uveitis" was discussed by 12 speakers (two from the United States); chairmen, Prof. J. Valdeavellano (Lima) and Prof. E. Palomino Dena (Mexico). And in still another hall, the subject of corneal conditions was given by six speakers (one from the United States). Following this, a meeting of the general assembly of the International Association for the Prevention of Blindness took place.

That evening a folklore performance by

the Society of the Ommegang took place in the Théâtre Royal de la Monnaie, especially presented in honor of the congress. This consisted of a pageant, procession, flag dancing, poetry, music, and ballet, in medieval costume and scenery, put on by members of the society, descendants of the old and noble families of Belgium, who usually do this once a year in private. It was an exceptional treat to view and to lose oneself for a short time in a most colorful period of history.

On Wednesday, September 10th, the main topic was "Senescence in ophthalmology," under the chairmanship of Sir Tudor-Thomas (Cardiff). The opening speakers were M. Bürger (Leipzig), J. François (Ghent), and G. Jayle (Marseilles). Thirty-six papers were scheduled in discussion (six from the United States). Obviously this was too many for the time allotted and many of the papers were read by title only, a most difficult decision for the chairman to make.

In the afternoon, a symposium on "Electroretinography," under the chairmanship of Prof. A. Franceschetti (Geneva) was held. Nine opening papers were scheduled (one from the United States); 19 free papers in discussion were on the program. In other halls, the subject of "Ocular optics," chairmen, Prof. G. Cosmetatos (Athens) and Prof. H. D. Dastoor (Bombay) was on the schedule. Thirteen papers (five from the United States) were read; and "Intraocular tumors and detachment of the retina," chairmen, Prof. M. Vannas (Helsinki) and Dr. L. B. Somerville-Large (Dublin) was the subject in another symposium. Thirteen papers were given (three from the United States).

In the meantime, in Hall E, the International Organization against Trachoma held its meeting under the chairmanship of G. B. Bietti (Rome). This meeting is historical, for reports were given confirming the discovery, for sure, of the virus of trachoma by Collier and Sowa (*Lancet*, 1:993, 1958). Dr. L. H. Collier of London gave an exciting paper, "Observations on trachoma virus isolated in embryonated eggs." Koch's postulates were amply fulfilled. It will be recalled by those who have read the interesting little book, *A Century of International Ophthalmology*, by Sir Stewart Duke-Elder and distributed at the time of registration to each of the members and delegates to the congress, that one of the main reasons for calling the first congress in 1857 was to discuss the baffling and widespread ocular disease called "military ophthalmia," which was trachoma and its bacterial complications emanating from Egypt. Duke-Elder says of this first congress "most of the time was devoted to the serious question of pandemic 'military ophthalmic,' but little of practical value emerged." Although it has taken more than a hundred years to determine the certain etiology of the widespread affliction, it seems eminently right that *finis* should be written on this subject at our centennial congress. Sixteen other papers on trachoma by world authorities were also given.

Wednesday night had no planned or formal social activities and most of the members and their guests headed for the World's Exposition for an evening of relaxation, serious or otherwise.

The next morning (September 11th), there was a symposium on "The etiology of cataract," chairmen, Prof. J. Nordmann (Strasbourg) and Dr. L. von Sallmann (United States). There were 18 opening speakers and 14 official discussors (eight in all from the United States). At the same time, the subject of "Glaucoma" was discussed in 12 papers in Hall C, chairmen, Prof. G. B. Bietti (Rome) and Prof. G. Karpe (Stockholm). Five members from the United States took part in this program. Likewise in Hall D, 12 papers on "Heredity and squint" were given; chairmen, Prof. M. Sobhy (Cairo) and Dr. S. Cooper (Bombay). Two members from the United States took part.

In the afternoon in Hall C, there were 12 free papers on "Glaucoma," chairmen, Professor de Ocampo (Manila) and Professor Sadoughi (Teheran). Three colleagues from the United States took part. In Hall D, 14 papers pertaining to "Ocular surgery" were

presented, five by members from the United States. The chairmen were Prof. R. Rodrigues Barrios (Montevideo) and Prof. Daeng Kanchanaranya (Bankok). A meeting of the International Professional Association of Ophthalmologists, under the chairmanship of Dr. Ch. Coutela, took place in Hall E.

That evening, the official banquet was held in the beautiful hall of La Madeleine, rue Duquesnoy. It was a brilliant event of great beauty, good food, delicious vintages, short speeches, lovely ladies, and rich appointments. Dancing, for those who survived, continued into the early hours.

The last scientific sessions took place Friday morning. Perhaps one of the most important meetings of the congress took place in the main hall when the subject "The preventive treatment of idiopathic and secondary retinal detachment" was discussed under the chairmanship of Prof. J. H. M. Weve (Utrecht). There were 16 opening speakers (three from the United States) and 15 official discussors (two from the United States). In Hall C, 12 free papers on "Experimental ophthalmology and biochemistry" were given (five by colleagues from the United States), chairmen, Prof. I. Michaelson (Jerusalem) and Prof. Marin Amat (Madrid). In Hall D, 13 free papers on operations and methods of examination were scheduled (one from the United States). The chairmen were Dr. N. Moura Brasil do Amaral (Rio de Janeiro) and Prof. W. Arkin (Warsaw).

During the hours of the formal exposition of scientific papers and throughout the meeting, 68 motion picture films, mainly on ophthalmic surgery, were shown in Hall B; 28 of these were by members from the United States. The hall was always crowded and the films were popular, as judged by the attention paid by the audience. It revealed that the lure of surgical motion picture films, like love, is universal, and needs no interpretation.

There were 51 scientific exhibits, some of them remarkably good. The exhibitors were from all parts of the world, 13 were from the United States. Fifty-four commercial firms, most of them European, had exhibits of interest and were always busy.

The final ceremonies took place on Friday afternoon. Closing speeches were given by representatives of the continents: Prof. A. Pillat (Vienna), Europe; Dr. A. E. MacDonald (Toronto), North America; Prof. Nguyen Dinh Cat (Vietnam), Asia; Prof. Courtis (Buenos Aires), South America; Prof. Mohsen Soliman (Cairo), Africa; and Dr. J. Bignell (Melbourne), Australasia. Then followed the closing speeches of the president of the International Council, Sir Stewart Duke-Elder; general secretary of the congress, Prof. Jules François; and the president of the congress, Prof. Léon Coppez.

The congress officially adjourned at three p.m., but many of the members, their families, and guests then travelled by bus for an unforgettable evening in the beautiful city of Bruges. Here we did sight seeing, followed by a delightful and most informal supper of cold cuts, and so forth, and delicious beer, served in souvenir steins, in the ancient Pandhalle of the Halles (belfry) or Town Tall (14th century) of tourist delight. The servitors and members of the brass band were in 16th century costume and looked as if they had just stepped out of a Brueghel painting.

So much for a cursory diary of this historic congress. Naturally, one's personal impressions of the occasion will vary a great deal from those of the other fellow, depending on his accommodations (some good, some bad), the time spent in the scientific meetings, or with boon companions, or at the "Expo.," the renewal of world friendships or the making of new ones, in cafés or restaurants, and so on. All, however, will agree that it was an exceedingly well planned and executed affair, full of great interest, much beauty and pleasure, with many sparkling moments that will forever remain in our memories.

We shall ever be grateful, too, for the

splendid work of the executive committee, the president, Léon Coppez, the vice-president, Prof. Appelmans, the treasurer, Prof. R. Weekers, and, particularly, Jules François, who was never tired, always pleasant and unruffled, with a prodigious memory for minute details and a skilful hand for execution. Our tribute is due, too, to the organizing committee: P. Danis, L. Alaerts, J. Michiels, J. Kluyskeus, R. Hermans, Ch. Stroobants, J. Zanen, J. Wibo, Else Van de Briel, P. Le Grand, A. de Jaeger, and to the ladies committee, especially Madame Coppez, the president, and Madame François, the secretary, gracious and lovely, each of them.

It is extraordinary that the few ophthalmologists in such a small country as Belgium were able to put on a congress that ranks in every way with the best we have ever had. We ophthalmologists from everywhere salute and honor them for a fine job.

Our thanks are due, too, to *Excerpta Medica*, the International Medical Abstracting Service, for printing and distributing in plenty of time, abstracts from the 146 scientific papers, in French, German, and English.

The simultaneous translations in the three official languages of the papers that were read in the main hall, were exceedingly well done and did much to improve the interest of the contributions. In the other halls, it was impossible to have this done, and the old bugaboo of language difficulties was ever present. This was not too serious, however.

The next (XIXth) International Congress of Ophthalmology will be held in New Delhi, India, in January, 1962, so start to get your papers, films, and exhibits read, and see your travel agent, for it will be an event you will not want to miss.

Derrick Vail.

THE 1958 ACADEMY MEETING

The 63rd annual session of the American Academy of Ophthalmology and Otolaryngology was held in the Palmer House, Chicago, October 12 to 17, 1958. This was one of the largest meetings, if not the largest, ever held. A total of 6,014 members and guests were registered. Due to the efficiency of the secretaries under the direction of William L. Benedict and the familiar physical surroundings, the meetings, instruction hours, and so forth ran smoothly in their allotted and well-oiled grooves and, as usual, there was something for everyone every minute of an 18-hour day.

The teachers' sections held a combined meeting on the morning of the opening day, with the theme, "Are we good teachers?" discussed by F. L. Lederer, L. K. Frank, Ray N. Lowe, R. B. Lewis, and A. C. Furstenberg, past president of the academy. It was a fascinating and most valuable program of methods of instruction and the demonstration of a host of tried and proven audiovisual aids, many of which are unfamiliar to most of the members of the audience, by professional experts in these fields. It is hoped that these talks and presentations will be published.

On Monday, October 13th, the Joint Scientific Session filled the large ballroom to overflowing. The president, LeRoy A. Schall of Boston, in his opening address, chose as his theme, "Science knows no frontiers." In this excellent talk, he emphasized the brotherhood of scientific men everywhere. The guest-of-honor was Louis H. Clerf, now of St. Petersburg, Florida, whose address, "Retirement," was a classic on the gentle art of graceful retirement, full of quiet wit and humor and particularly great good sense. The prolonged applause at its conclusion was testimony to the full enjoyment the address afforded the audience. Following the presentation of 21 honor awards, a symposium on "The care of the surgical patient," was held. H. Walter Jones, Jr., of Boston, spoke on the "Preoperative evaluation of the patient"; W. H. Hamilton of Iowa City on "Some fallacies in choice of anesthetic agents"; and Alton Ochsner of New Orleans, on "Postoperative surgical care." These talks by invited guest speakers, physicians and surgeon,

were highly instructive and of interest to all members.

The Scientific Section on Ophthalmology held its session thereafter each morning, as usual, through October 16th. The presiding officers were the vice-presidents, F. Bruce Fralick of Ann Arbor, Michigan, and Philip M. Lewis of Memphis, Tennessee. The program consisted of clinicopathologic case reports, scientific motion pictures, and eight major papers of the usual high standard imposed by the academy. A symposium on "Ocular effects of ionizing radiation and radiotherapy" occupied the session of October 15th. The basic concepts were discussed by D. G. Cogan, Titus C. Evans, and Ludwig von Sallmann. The clinical application was discussed by M. W. Schulz who spoke on "Radiotherapy of ocular adnexa"; A. W. Forrest, "Radiotherapy of ocular lesions by X rays and gamma rays"; J. E. McDonald and F. M. Wilson, "Radiotherapy of ocular lesions by beta particles"; and W. F. Hughes, Jr., on "Radiotherapy compared to other forms of ocular therapy." The papers were factual and conservative, with emphasis placed on the inherent dangers to patient and physician in the use of these agents, and timely warnings that they should not be used by those who are not experts and should not be employed unless other forms of treatment fail.

The XV Jackson Memorial Lecture was given on October 14th by Arthur Linksz of New York. He spoke on "Aniseikonia" with particular regard to the Jackson-Lancaster controversy. The exposition of this difficult subject was indeed most lucid and graceful and all of us have a better understanding of this thorny topic as a result. The lecturer was warmly applauded.

Dr. Fralick, the chairman, graciously gave permission for the showing of three unscheduled films on the use of alpha-chymotrypsin to produce zonulolysis, a subject of current interest and excitement in cataract surgery. The first of these was by J. Barraquer of Barcelona, Spain, who was the first to introduce the use of this extraordinary drug to ophthalmologists in May, 1958. His motion picture, discussed by Derrick Vail, was most convincing and was enthusiastically received. Olga Ferrer, of Cuba, then showed a motion picture of two of her own cases, both well done and successful, and Richard C. Troutman of New York contributed a film on the use of this enzyme in congenital cataract surgery. This was most valuable, for it showed by dramatic and frightening loss of vitreous the grave hazards of attempting to perform intracapsular cataract surgery in these cases. All speakers condemned in no uncertain terms such surgery for congenital cataracts in children and young adults.

Another high spot of the scientific program was the paper by G. Meyer-Schwickerath of Bonn, West Germany, on the "Indications and limitations of light-coagulation of retinal lesions." His apparatus and work in this field are, of course, widely known by now, but the members were most appreciative of the opportunity to hear and see this modest young man whom we all admire and respect.

Other high spots were the papers by A. B. Reese and G. W. Cleasby on "The treatment of iris melanoma," and G. Fonda and D. Snydacker on "Optical aids for low visual acuity."

The Section on Instruction in Ophthalmology provided us with 145 individual and 53 continuous courses covering every field in ophthalmology. As usual, these courses were almost all completely sold out. Two hundred and forty-five instructors, fellow members, gave 373 hours of instruction, a most remarkable showing.

The usual special scientific programs (open meetings) filled in what few gaps there were in time. These were (1) the meeting of the American Orthoptic Council and American Association of Orthoptic Technicians at which was held a symposium on "Office orthoptics by the ophthalmologist." Taking part in the discussion were W. P.

Chamberlain, Florence M. MacLean, J. P. Cowen, and Frances Fowler. (2) The meeting of the Committee on Reconstructive Plastic Surgery, with the presentation of a symposium on "Ptosis complications." Taking part were C. E. Iliff, F. Bruce Fralick, R. N. Berke, J. S. Crawford, and R. A. Schimek. (3) The meeting of the American Society of Ophthalmologic and Otolaryngologic Allergy, at which F. C. Blodi discussed "Sympathetic ophthalmia as an allergic phenomenon." All meetings were well attended and obviously popular.

A real tragedy occurred on the night of October 15th, when the rooms containing the scientific exhibits caught fire which completely destroyed every single exhibit, some of which represented years of labor to assemble and most of which can never be replaced, such as the numerous and beautiful water-color paintings of diseases of the macula by Bertha Klien.

There were 24 of these exhibits, 11 of which were ophthalmologic. In this field, the first ribbon was awarded to Paul C. Cibis, T. Yamashito, and W. Moor of Saint Louis, for their exhibit, "Siderosis and hemosiderosis oculi: An experimental and clinical study." The second ribbon went to the Armed Forces Institute of Pathology, Washington, D.C., represented by L. P. Ambrogi on "Processing the whole eye in paraffin." The third was awarded to Bertha A. Klien, Chicago, for her splendid exhibit, "Diseases of the macula: Basic and pathologic changes modifying the clinical picture"—small recompense for the irrevocable loss of her material.

The usual scheduled social events were thoroughly enjoyed. These were the president's reception on the opening day, the banquet and the cocktail and dinner meetings of 38 alumni groups of various universities, hospitals, and so forth. The Illinois Eye and Ear Infirmary held a special dinner as a centennial celebration, and this was indeed a feature.

As is also customary, innumerable groups of friends held informal reunions, sessions, and celebrations at odd hours. It is hoped that each member has caught up with his sleep by this time, for the week was a strenuous, exciting, and happy one for all.

The officers elected for the ensuing year are: President, John H. Dunnington of New York; president-elect, Dean M. Lierle of Iowa City; first vice-president, Gordon D. Hoople of Syracuse, New York; second vice-president, S. Rodman Irvine of Beverly Hills, California; third vice-president, Lyle M. Sellers of Dallas, Texas; and to the council, E. B. Dunphy of Boston, Massachusetts. The next meeting will be held October 11 to 16, 1959, at the Palmer House, Chicago.

Derrick Vail.

THE JAPANESE OPHTHALMOLOGICAL SOCIETY

One of the most pleasant experiences I have ever had was a recent trip to Japan as guest of the Japanese Ophthalmological Society. Although it was of only a week's duration, the visit will certainly stand out as a very bright spot in my memorabilia. The distance is no longer a great deterring factor, even for short visits—the route of the Northwest Airlines via Alaska and the Aleutian Islands has made the traveling time surprisingly short.

The Japanese Ophthalmological Society has a membership of 2,050 and at the recent 62nd meeting, held in Niigata City, there were 1,024 members in attendance. It is their custom to hold this meeting annually for three days in April or May. The president this year was Prof. Masakichi Mikuni, the incumbent of the Chair of Ophthalmology at the University of Niigata.

The meeting was one of the best organized and executed that I have ever attended. The papers were given with dispatch, and the allotted time of 10 minutes was closely adhered to. The projection was excellent. In the center of the auditorium there was

double projection and, in addition to this, single projection on each side. Across the center aisle of the auditorium there were four microphones with an attendant at each. These were used by the discussors who spoke from their seats into the microphones which were brought them immediately by the attendant. This enabled many discussors to comment with a minimum loss of time. At the close of each discussion another attendant would go immediately to the doctor who had spoken for his name and address. Another feature of the projection, which seemed to be a good one, was the long runners which accommodated 10 two by two slides so that the speakers could arrange their slides themselves in the proper order and the projectionist then merely put the runners through the machine. This prevented any confusion about the order in which the slides were to be shown and also saved time in changing slides when a rapid succession of them was called for.

Ninety-four papers were presented at this meeting in abstract form, 10 minutes being allotted to each author. In spite of the fact that only 15 of these papers were of a clinical nature, the attendance at each session was amazingly good, averaging around 600. I doubt that a program devoted essentially to basic research work would attract such a large number of ophthalmologists in our country. Each paper was discussed in the manner previously mentioned. The experimental studies revealed a wide range of interest in biochemistry, electroretinography, and electromicroscopy. Biochemical approach seems to be a dominant theme in Japanese research. There were eight papers treating the eye as a whole, but biochemical techniques were used in some 32 of the 94 papers.

In Japan, after graduation in medicine, those who train for ophthalmology work for their Ph.D. degree for four years in a university eye clinic. Part of this period is spent in research on which a thesis is presented for a Ph.D. degree. Many of the papers presented at the Japanese Ophthalmological Society are reports on such research. This accounts for the great number of papers on basic work.

It is of interest to see some of the diseases occurring more frequently in Japan than in our country. Some of these bear the names of the Japanese ophthalmologists who first recognized and described them. Arrangements were made for me to see examples of Oguchi's disease and Vogt-Koyanagi-Harada disease. It was noteworthy that Behçet's disease is relatively common in Japan. At the clinic of the School of Medicine of the University of Tokyo they listed 69 cases (0.35 percent of the cases seen for the year 1955). Trachoma, of course, is common and this same clinic listed for 1955, 557 cases or 2.86 percent of the total. Central angiospastic retinopathy seems to be more common in Japan than in our country and the incidence was particularly high during the war. In the eye clinic of the University of Tokyo, in 1942, the incidence was 1.24 percent of the total while in 1955 it was only 0.56 percent (approximately the same as it was in 1935). This seems to indicate that there is a stress factor in this disease. Malignant melanoma of the uvea is an extremely rare occurrence, there being only 70 cases reported in the Japanese literature. A disease which was of great interest to me was Takayasu disease (pulseless disease), and at the University of Tokyo's clinic they listed for 1955 seven such cases, or 0.04 percent of the total.

In 1908 Takayasu reported the first case with a complicated cataract and unusual ocular findings, and, at the same meeting, Onishi and Kagoshima reported two similar cases with the added observation of absent radial pulses. It was apparently not until 1939 that absence of pulsations in the common carotid arteries was reported as part of the syndrome.

While the absence of pulses in the arms and neck is the hallmark of "pulseless disease," there are many other conditions which may produce a similar phenomenon. This is, perhaps, best called the "aortic arch syn-

drome," and Ross and McKusick, reviewing 100 cases in 1953, divided the cases into 12 groups, according to their presumed etiology.

It becomes evident that the name "Takayasu's disease," or its synonym "pulseless disease," should be reserved for cases of acquired nonspecific arteritis which usually occurs in young women.

Visual disturbances are the most constant manifestation of the disease and the chief ocular symptom is transient loss of vision, usually episodic and lasting a few seconds or several minutes at first, followed in many cases by permanent visual loss, even to the point of total blindness. The obscuration is characteristically brought on by a change in position, rising to an upright position or raising the head, and vision returns promptly when the head is lowered or when the individual lies down.

Objective ocular signs include the progressive formation of peripapillary arteriovenous anastomoses, lowered retinal arterial pressure, retinal hemorrhages, and peculiar slowing of the circulation so that the blood elements may be seen moving in segments through the vessels of the retina. Unfortunately cataracts (usually nuclear but occasionally complicated) frequently develop to obscure the retinal findings. In the anterior segment iris atrophy, mydriasis, and hyperemia of the conjunctiva and sclera seem to be common signs.

Most of the early cases were reported in the Japanese literature but, in recent years, instances of this condition have appeared in various parts of the world.

It would appear that there have been six cases reported in the American ophthalmic literature. These have been by Caccamise (1954), Lawson (1954), Pinkham (1955), Figley (1956), and Ostler (1957).

Many Americans have either visited or lived in Japan and have, therefore, had the opportunity to form their own opinions of the country and the people. I have never heard of anyone who has been to this country who did not leave it with an affection for the Japanese and a respect for their country. I certainly belong in this category. Like others, I found the Japanese to be a people of vitality combined with serenity. Their great accomplishments are exhibited with modesty. They seem to have respect for law and order and a feeling for the eternal fitness of things.

The social side of the meeting was delightful. The Japanese doctors and their wives could not have been more hospitable and cordial. There were two official banquets, one in Japanese style and the other in Western. At each the pretty geisha girls dispensed their graciousness. And so, may I say, Japan is not far away as measured in time and you will find it a fascinating country and the people charming. Sayonara.

Algernon B. Reese.

REFERENCES

Caccamise, W. C., and Okuda, K.: Takayasu's or pulseless disease. Am. J. Ophth., **37**:784-786, 1954.

Caccamise, W. C., and Whitman, J. F.: Pulseless disease: A preliminary report. Am. Heart J., **44**: 629-633, 1952.

Figley, M.: New contributions of radiology to ophthalmology and otolaryngology through vascular visualization. Tr. Am. Acad. Ophth., **60**:768-773, 1956.

Lawson, L. J., Jr.: Pulseless disease. Am. J. Ophth., **37**:788-790, 1954.

Ostler, H. B.: Pulseless disease (Takayasu's disease). Am. J. Ophth., **43**:583-589, 1957.

Pinkham, R. A.: The ocular manifestations of the pulseless syndrome. Acta XVII Cong. Ophth. 1954 (Canada-U.S.A.) 1955, pp. 348-366.

II. INTERNATIONAL COURSE OF OPHTHALMOLOGY

INSTITUTO BARRAQUER

Almost 200 ophthalmologists from 33 countries attended the second postgraduate course in ophthalmology established by the Barraquer Institute in Barcelona, Spain, from September 16 to October 1, 1958. Forty-four well-known international experts in various subjects in ophthalmology gave lectures, discussions, and film demonstrations. The instructors came from 11 countries outside of Spain, as follows: United

States of America, 12; France, two; Brazil, three; Argentina, three; Great Britain, six; Italy, six; Belgium, one; Panama, one; West Germany, five; Cuba, one; and Switzerland, three.

Symposiums were held on glaucoma, keratoplasty, lens surgery, intraocular plastic lenses, detachment of the retina, and strabismus. In addition, 18 papers on miscellaneous topics were given.

The official languages were French, Spanish, Italian, German, and English. Simultaneous translations through ingenious and effective transistor radio receivers were provided to all participants and, on the whole, were satisfactory. The lively and valuable discussions that concluded each symposium were given by selected panel members.

In addition to the scientific program, each day many excellent surgical films were shown, and hospital rounds and visits to the out-patient clinics were made. Surgical clinics were put on in the afternoons and early evenings by Drs. Ignacio Barraquer, Alfredo Muiños Simon, Joaquin Barraquer, and Pascual Gonzales-Marin. This was an attractive and most popular feature. For those who were unable to get into the operating theater, a closed-circuit television of each operation was provided. In many ways, viewing the operation on television was more satisfactory than watching the actual operation.

The most interesting and exciting event was the demonstration, by actual surgery and film, of the action of alpha-chymotrypsin on the zonular ligaments of the lens (zonulolysis). It will be recalled that Joaquin Barraquer, a superb ophthalmic surgeon, announced the great discovery of the lytic effect of chymotrypsin on the zonule in May, 1958. Since then he has used it in more than 200 cases of cataract surgery, with excellent results. He demonstrated to our satisfaction that the enzyme does not injure the hyaloid membrane, nor, insofar as is now known, the ciliary epithelium. There is no doubt about the forward dislocation of the lens and for that reason, while the use of the enzyme solves some of the problems of cataract surgery (for example, resistant zonular fibers) it raises some others (for example, difficulty in grasping the lens, particularly with forceps, and extracting it without damage to the corneal endothelium or of pushing it back into the vitreous). The use of the enzyme to facilitate the removal of the congenital cataract in its capsule in an infant or child is only effective in so far as the zonulolysis is concerned. It does not dissolve the embryonic adhesions of the vitreous-lens (mesoblastic remnants of the tunica vasculosa and also the ligamentum hyaloidea capsulare) that are probably present in a high percentage of individuals under the age of 20 years, and decreasing with age. For this reason loss of vitreous is very apt to occur, as indeed it did in several of Barraquer's patients. Therefore, in my opinion, the intracapsular extraction of the lens in children should be strongly condemned.

The social events that accompanied the course were outstanding and delightful. The hospitality of the Barraquers was most generous, gracious, and lavish.

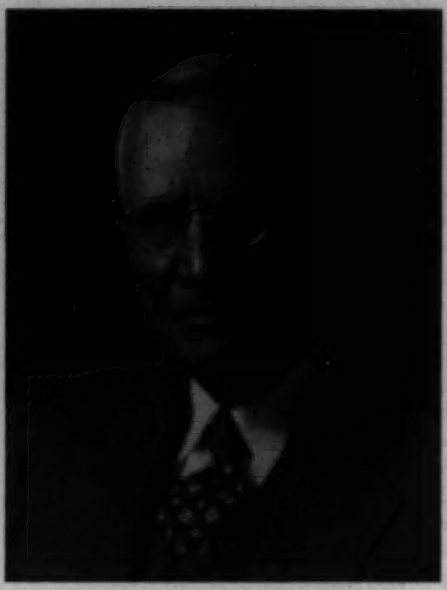
Those who attended the course showed unusual interest and devoted attention, crowding into the small auditorium and overflowing into the corridors in spite of fatigue, heat, long hours, and some physical discomfort, and staying until the very end. This was an obvious tribute to the excellence of the course and the skill with which it was organized and administered.

Derrick Vail.

OBITUARY

CHARLES A. THIGPEN, M.D.
(1865-1958)

As he had been doing for many of his 92 years, Dr. Charles Alston Thigpen enjoyed his weekly horseback ride on Wednesday at "Cedar Creek," his plantation near Greenville, Alabama. Also, as was his custom, on Saturday, he saw a dozen consultation pa-



CHARLES A. THIGPEN, M.D.

tients in his office in Montgomery, Alabama. Since his "retirement" on October 26, 1957, the 65th anniversary of his entry into the practice of medicine, he made no charges though he worked more diligently than ever on his patients, many of whom were old friends.

On Monday he developed a slight cold, and on Tuesday he had a mild heart attack, his first. His cautious internist insisted on staying at his home until late to watch over him and during the evening inadvertently reached for a cigarette. Dr. Thigpen, who detested smoking, said to his internist with a smile, "If you would listen to me and stop smoking, you would live a lot longer!"

The next morning, April 23, 1958, after talking pleasantly to his startled family who were gathered around because his condition had become worse, he closed his eyes and departed from this world with all of the graciousness and dignity with which he had lived. So ended the epic life that had begun near Greenville in the year of the cessation of armed hostilities between the States.

Growing up through the ordeal of Reconstruction when the South was an occupied country, he resented Yankees, although he excepted from this judgement all Northeners he came to know. As time went by this prejudice, more symbolic than actual, mellowed. However, when a "Northener" failed to live up to his responsibilities in life, Dr. Thigpen would smilingly say, "Well, what could you expect from a Yankee anyway?"

At the age of 20 years, he received the A.B. degree from Howard College, then at Marion, Alabama, and two years later in May, 1888, received the Doctor of Medicine degree from Tulane Medical School. Dr. Rudolph Matas was his anatomy teacher and lifelong friend. He then went into general practice with his father in Greenville for one year. Dr. Thigpen referred to this year as the one in which he learned most, because he discovered how little he knew that he wanted to know about medicine. He decided to become an eye, ear, nose, and throat specialist, and interned at the New Orleans Eye, Ear, Nose, and Throat Hospital 1888-1890. Then he spent two years in New York, London, Vienna, and Heidelberg studying under Parsons, Lister, Morrell McKenzie, and others. In October, 1893, he became associated with Dr. B. J. Baldwin of Montgomery, Alabama, in the specialty of eye, ear, nose, and throat, and two years later Dr. Baldwin retired because of ill health.

Dr. Thigpen was the first ophthalmologist in the south to have had such advanced training and his success was immediate. Yet he did not live on past accomplishments but constantly advanced with the new discoveries in medicine.

Realizing that bronchoscopy was needed in the southeast, in 1911 he went to Frieburg, Germany, and spent several months in learning this new specialty. He purchased the equipment necessary and was the first doctor in the south to perform this service.

In World War I he was commissioned a major and served as the chief of the Ophthalmology Section of the Base Hospital at

Fortress Monroe at Old Point Comfort, Virginia. In 1895, he was one of the three founders of the American Society of Otology, Rhinology, and Laryngology. In 1953 this society had him as their guest-of-honor at their annual meeting; the other two founders having long since passed on.

In 1934, the University of Alabama awarded him the degree of LL.D. and, in 1936, he was elected president of the Alabama State Medical Association. He was a member of the State Board of Censors from 1932 to 1945.

In 1952 he was given the Award of Merit by the Alumni Association of the New Orleans Eye, Ear, Nose, and Throat Hospital.

In addition to membership in the county and state medical societies, the Southern Medical Association, and the A.M.A., Dr. Thigpen belonged to the American Academy of Ophthalmology and Otolaryngology, the American Association for Research in Ophthalmology, American College of Surgeons, and he was a commissioner for the Conservation Division of the Society for the Prevention of Deafness.

He was a trustee of the Eye Foundation, Inc., the Alabama School for the Blind, and the Peter Bryce Insane Hospital.

In 1896, he was married to Daisy Lee Bissell of Charleston, South Carolina. They had three children, all of whom survive: Dorothy (Mrs. Edmund B. Shea) of Milwaukee, Wisconsin, Elizabeth (Mrs. Wiley C. Hill, Jr.), and Charles A. Thigpen, Jr., both of Montgomery, Alabama. Dr. Thigpen's devotion to his family, which they returned in full measure, was reflected in the happiness and serenity of his life. When Mrs. Thigpen died in 1947, Elizabeth, who with her family lived a mile away, came to manage his household and have breakfast with him every morning until his death. Dr. Thigpen's greatest interest after his family and his patients was in hunting, and he kept a large number of dogs and horses. He required that his guests follow the same strict code of sportsmanship that he did.

Probably the most famous of the innumerable legends about Dr. Thigpen was that which dealt with his charge for an operation in 1921 on Mr. W. C. Bradley, then chairman of the Board of the Coca-Cola Company. Dr. Thigpen, was successful in relieving Mr. Bradley of excruciating headaches of several years' duration by opening a left sphenoidal sinus filled with pus which had received no attention previously, although some nasal polyps had been removed and different types of nose drops recommended. When he recovered and expressed his appreciation profusely, Dr. Thigpen sent him a bill for $250.00. The bill was returned with a check for $1,000.00. Dr. Thigpen sent back the check, and insisted only on the $250.00. Since he would not accept the larger fee, Mr. Bradley then urged Dr. Thigpen to buy Coca-Cola stock, which he did. Perhaps Mr. Bradley knew that a stock split was in the offing; at any rate, the stock was split and soon bid up so that it had doubled in value in a few weeks; the investment continued to increase in value throughout Dr. Thigpen's life.

Dr. Thigpen was fearless in speaking the truth, even though it sometimes resulted in embarrassment. Three decades before the sulfas and antibiotics, when vaccines were just being developed, Dr. Thigpen saw as he passed down a hospital hallway that the son of an old acquaintance was weakening rapidly from osteomyelitis. Dr. Thigpen stopped the father and expressed his sympathy for what appeared to be a fateful outcome. The father asked Dr. Thigpen what he would do if it were his own son, and Dr. Thigpen said, "Why, I would take him away from the doctor you have and this hospital, and I would go to another city and there see Dr. Skinner who knows about these new vaccines." The father immediately did this, a vaccine was developed and used, and the boy recovered.

At the age of 91 years, Dr. Thigpen performed an enucleation without difficulty under unusual circumstances. The patient was an old friend of Dr. Thigpen's. One of his

eyes had disintegrated from absolute glaucoma and was very painful. He refused hospitalization and begged Dr. Thigpen to remove his eye, reminding him that 65 years previously he had performed many operations at patient's homes on kitchen tables. Dr. Thigpen thereupon returned to his office, sterilized his enucleation instruments, wrapped them in a sterile towel and took them to the man's home. He injected the orbit with Xylocaine, performed a near painless removal of the eye, and sutured Tenon's capsule and the conjunctiva together after the bleeding stopped. Few ophthalmologists have performed successful eye surgery at this stage of life.

Like most doctors, Dr. Thigpen never turned away a patient because he had no money. However, unlike many doctors, he often paid the hospital bill besides giving his services free when no funds were available in serious cases.

Yet it was not his incomparable gifts of mind nor his brilliant scientific skill which lifted him above the rank and file of physicians. As was said of Sir William Osler: "He advanced the science of medicine . . . yet individually he had a greater power. He became the friend of all he met. He knew the workings of the human heart, metaphorically as well as physically. He joyed with the joys and wept with the sorrows of the humblest. He stooped to lift them up to the place of his royal friendship, and the magic touchstone of his personality helped many a desponder in the rugged paths of life."

Alston Callahan.

CORRESPONDENCE

GLAUCOMA SURVEY IN A SMALL COMMUNITY

A glaucoma survey was held in the city of Missoula, Montana, on May 24, 1958. This survey was done with the co-operation of the local ophthalmologists under the auspices of the Delta Gamma Alumni Society of Montana State University. The survey was staged with the co-operation of the National Society for the Prevention of Blindness. Missoula is a city of approximately 30,000 inhabitants, with a comparatively large outlying population. There are three general hospitals in the city and 60 physicians.

A total of 301 persons reported at the clinic for screening. These ranged in age from 20 to 89 years. The procedure was to record the name, address, and age of the patient. Then the vision was taken, both for far and near. The tension was measured with a Schiøtz tonometer and, if the tension was 20 mm. Hg or more (new scale), a visual field was done with the Harrington flash field recorder. The patients were classified as follows:

1. Normal findings	184
2. Increased tension	22
3. Defective vision	54
4. Defective fields	17
5. Borderline cases	21

The results were tabulated and all persons except those in the normal category were sent a slip requesting them to see the ophthalmologist of their choice for further examination.

Fifty-four cards were sent out; 32 of those contacted consulted ophthalmologists in Missoula. One patient came to Missoula because she had read of the survey and the description of the symptoms of glaucoma. She consulted a physician and was found to have chronic wide-angle glaucoma.

The total number of glaucoma cases found with this survey was six. Three of these were new cases, and three were already under treatment. This represents two percent of the total number of persons examined.

This was the first survey of its kind ever attempted in western Montana. The very fact that, starting from scratch, it was possible to interest over 300 persons augurs well for the continuation of this effort. Even though the percentage of glaucoma cases found does not seem high, the interest shown by the persons who attended the survey has

fostered a growing awareness of this disease and its symptoms.

The Delta Gamma Alumni Society was headed by Mrs. Fred H. Lowe, the wife of a local physician. It is planned to hold the survey yearly.

(Signed) H. R. Crisman, M.D.
Wendell L. Jones, M.D.
Edward S. Murphy, M.D.
Geo. G. Sale, M.D.
Patricia A. Murphy, M.D.

BOOK REVIEWS

A Century of International Ophthalmology (1857-1957). By Sir Stewart Duke-Elder. London, Kimpton, 1958. 91 pages, 16 illustrations, appendix. Price: Not listed.

This is a delightful history of the international congresses of ophthalmology from I through XVII, 1857 to 1957, written by the old Maestro. We learn that the Congress of Ophthalmology is the oldest surviving one of any of the medical sciences and thus is another "first." International congresses are a dime a dozen these days but it is nice to know that we have paved the way in this as well as in other ventures (for example, American Boards, special journals, special societies, etc. etc., —ho! hum!)

The first congress met in Brussels, 1857, primarily to discuss the ophthalmoscope and the new world that is revealed and "military ophthalmia" (trachoma) that had turned out to be a scourge. The last congress also held in Brussels (1958) still had papers on ophthalmoscopic problems but also wrote *finis* to the problem of the etiology of trachoma.

In between, the congresses, held in many important cities, chiefly in Europe, met as often as the rumors of war, or actual war, permitted. Some were good, very good from a scientific view point; others, not so good. The attendance and interest have grown from 150 in 1857 to over 2,000 in 1958. The number of papers presented developed from very few to probably too many. But, in any event, the good fellowship and friendships engendered, the informal discussions over beer, wine, or coffee, and the social activities, often glittering, have remained unchanged, fortunately, through this century.

You expect this little book to be entertaining and informative and you are not disappointed. It should be read with great pride by all of us. Derrick Vail.

Actualites Latines d'Ophtalmologie. Edited by W. Duque Estrada and G. E. Jayle. Paris, Masson et Cie, 1958. 314 pages, table of contents. Price: Not listed.

This is apparently the first of a projected series of volumes whose title is perhaps best translated as "Recent Advances in Latin Ophthalmology." An impressive staff of contributors from South America and southern Europe form the editorial committee and international co-ordination is under the direction of those two famous Latins, Sir Stewart Duke-Elder and Derrick Vail.

The present volume is divided into eight parts. Of these the shortest is Part I on anesthesia in which Almeida and Ferreira make a plea for general anesthesia in cataract surgery. Part II on visual fields is the longest section and is in reality a monograph on the subject. There are five chapters in this section introduced by the French master perimetrist Dubois-Poulsen. The major portion of this section is devoted to mesopic visual fields. These fields are performed under conditions of intermediate illumination where there is maximum sensitivity of the cones and the rods are at the threshold of sensitization. Numerous case reports illustrating the advantages of this technique will be of interest to ophthalmologists in America where mesopic campimetry is as yet rarely used. In contrast the third section on glaucoma will be of less interest since it concerns gonioscopy, standardization of tonometers, traumatic hypertension, and other subjects well covered in the American literature.

In the fifth section on pathology there is an interesting article on conjunctival capillary fragility in diabetes. The authors show that in diabetics with retinopathy, the conjunctival capillaries are more easily ruptured by an applied suction cup. In these cases the author advises the use of vitamin C, vitamin P, and others, and concludes with proper conservatism "one can sometimes avoid the severe hemorrhagic complications of diabetic retinopathy."

An excellent experimental study on healing of the cataract wound is reported by Duque Estrada. He concludes on the basis of histologic examination of dogs' eyes at varying intervals after surgery that catgut produces far more reaction and infiltration than silk unless the suture is entirely corneal.

The final article illustrates the pitfalls inherent in clinical research and the athletic phenomenon of "jumping to conclusions." Twenty-four eyes (12 patients) with pigmentary degeneration received "retinal illumination" according to a specified technique. Of these 24 eyes, 10 showed no change, nine improved "one-tenth," and five improved "two-tenths." The author concludes that "light . . . is able to improve, in a notable number of cases . . . the visual acuity . . . in pigmentary degeneration of the retina." No comment.

David Shoch.

THERAPEUTIC HEAT. Edited by Sidney Licht, M.D. Baltimore, Waverly Press, 1958. 466 pages, 127 figures, chapter bibliographies, author and subject indices. Price: $12.00.

This is a symposium by 23 contributors. The jacket correctly states: "Here, under one cover, the reader will find authoritative discussions on the physics, biophysics, and physiology of heat, as well as descriptions of the apparatus and methods used in the hospital, office, and home."

Louis Daily and Ray K. Daily have written the chapter relating to diseases of the eye. All relevant material is reviewed. Infrared radiation is more effective than the heating pad in elevation of ocular temperature, and diathermy is more effective than either. Microwaves appear particularly suited to the eye since they can be brought to a focus by a metallic reflector. Unlike short waves, the microwaves do not produce an unpleasant sensation of heat when the lids are moist or covered with ointment. The authors properly emphasize that the applications of any form of heat are only supplemental to other and more specific therapeutic agents.

James E. Lebensohn.

THE RHINOGENOUS DISEASES OF THE ORBIT. By R. Herrmann, M.D., Tübingen. Fasc. 2 of Informal Treatises in the Field of Oto-Rhino-Laryngology. Stuttgart, George Thieme, 1958. 83 pages, 42 illustrations, bibliography. Price: $4.30.

Herrmann discusses the rhinogenous diseases of the orbit from the point of view of the otorhinolaryngologist. In three separate chapters he considers inflammatory processes, orbital complications resulting from mucoceles and tumors of the accessory sinus, and orbital complications due to injuries.

The fairly adequate descriptions of the clinical symptoms are accompanied by very well-reproduced illustrations of typical cases. Diagnostic problems and the discussion of the gross and histologic pathology are treated only in a vague and general manner. The same approach is evident as far as conservative and surgical therapy is concerned. However, such an attitude may be justified by the author's plea for close co-operation between the ophthalmologist and the otorhinolaryngologist, where only the latter should be familiar with technical details.

It would seem that the majority of the inflammatory conditions under discussion are mostly of historic interest. In this era of antibiotics, most of the diseases, if recognized early and treated adequately, have lost the

stigma of horror and fear formerly attached to them. This is attested by the author's observation of 6,000 patients with sinusitis in the clinic of Tübingen: only 32 cases showed involvement of the orbit or lids, and 15 of these responded to conservative treatment.

Only 10 percent of the some 200 titles appearing in the bibliography refer to non-German publications.

Stefan Van Wien.

PREVAILING PROBLEMS IN THE CROSS-EYED CHILD. By R. Brückner, M.D. Basle and Stuttgart, Benno Schwabe & Co., 1958. 68 pages, 17 illustrations. Price: sFr 6.00.

This brochure, written by an ophthalmologist, is addressed to the parents of cross-eyed children, to social workers, and to nursery and public school teachers. Be it stated that every ophthalmologist will profit greatly by reading this marvelous little volume from cover to cover, so that he may never lose sight of the psychologic problems of those "on the other side of the fence."

There is an erudite explanation for the correlation between accommodation and convergence, normal and abnormal correspondence, amblyopia (including its treatment by means of occlusion and pleoptics), and the surgical indications. It should become clear to every reader why each case of strabismus has to be approached individually, and why there is not one method as a cure-all in every instance.

Most of all, the parents are encouraged to co-operate with the physician and orthoptist. The mother who emphatically declares in the presence of the child that she will never agree to him wearing glasses cannot expect him to accept them once it has been established that glasses are a necessary part of the procedures to be carried out. The parent who shows his unhappiness over the prospect of prolonged occlusion of one eye will create an emotional problem in a child who otherwise would be perfectly adjusted to such a measure.

An excellent chapter deals with the behavior of the general public in contact with children who have to wear glasses or occlusion. Unfortunately there is no motive for most people to become interested in these problems.

Wouldn't it be a splendid idea to hand a copy of this pamphlet to the child's parents on the first visit? All the many questions which must be on their minds are anticipated and answered in much greater detail than would be possible in the surroundings of a busy office or clinic. The material is treated in a very general manner and does not in any way encourage the parents to start their own treatment or modify the treatment outlined by the physician or orthoptist.

Stefan Van Wien.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

1

ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

Dawson, H. **The blood vessels of the human optic chiasm and their relation to those of the hypophysis and hypothalamus.** Brain 81:207-217, 1958.

Dawson reports the anatomic studies of the arteries and veins referred to in the title of his report. The data are the result of microdissection which are made clear by description, drawings and beautiful photographs. (9 figures, 22 references)

Irwin E. Gaynon.

Gregersen, E. **The spongy structure of the human iris. Preliminary report.** Acta ophth. 36:522-535, 1958.

By intravital perfusion of the anterior chamber with dextran solutions before enucleation and histologic examination of the tissue, the spongy nature of the iris could be demonstrated; the mesodermal tissue of the iris becomes thoroughly saturated with the substance. (4 figures, 50 references)

John J. Stern.

Kato, Yoshiko. **A histologic study on the distribution of nerves in the human lacrimal gland.** Jap. J. Ophth. 2:103-108, May, 1958.

The nonmedullated nerve fibers subdivide and finally form a reticular termination surrounding the basal surfaces of the acini, excretory ducts, and fat cells. The medullated nerve fibers apparently end on the basal surface of the secretory portion. Other fibers lose their myelin sheaths, enter between the cells of the excretory duct, and terminate as intraepithelial fibers. Myoepithelial cells are apparently innervated only by the terminal reticulum. (10 figures, 10 references)

Irwin E. Gaynon.

Miyake, Y. **A histological study of ciliary muscle.** Acta Soc. Ophth. Japan 62:810-821, July, 1958.

The anatomy of the ciliary muscle was studied in human and animal eyes. Miyake concludes that Brücke's muscle has its origin mainly from the sclerocorneal trabeculum and partially from the scleral spur. The inner layer of the muscle reaches the vitreous membrane at the pars plana. The external layer of the muscle runs backwards to reach the sclera at the optic disc. (22 figures, 47 references)

Yukihiko Mitsui.

2

GENERAL PATHOLOGY, BACTERIOLOGY, IMMUNOLOGY

Cavalhero, J., Domingues, M. and Carvalho, C. **Experimental work in hypertensive retinopathy.** Rev. brasil. oftal. 17:249-257, June, 1958.

The authors review briefly the experimental work done in generalized hypertension and describe the ophthalmoscopic lesion observed in the malignant type of experimental hypertension: hemorrhages, exudates, retinal detachment, papilledema, white exudates. The ophthalmoscopic lesions observed in the benign form are: retinal edema, white exudates, hemorrhages and vascular changes. The authors believe that the best method to study these lesions is by stereobiomicroscopy, as this method makes possible the detection of superficial retinal edema, which is an early sign. (30 references)

Walter Mayer.

Gilkes, M. J., Smith, C. H. and Sowa, J. **Staining of the inclusion bodies of trachoma and inclusion conjunctivitis.** Brit. J. Ophth. 42:473-477, Aug., 1958.

A modification of the Rice method of staining smears from eyes with trachoma or inclusion conjunctivitis for inclusion bodies is described. By this means a diagnosis can be made with accuracy in approximately 15 minutes in contrast to the 60 or more minutes required by the well-known Giemsa staining technique. This method was particularly valuable in Gambia and Jordan where all study was made in the field. Scrapings are smeared on a clean dry slide and exposed to air for about five minutes. This is then covered with a 5 percent solution of iodine in 10 percent potassium iodide aqueous Lugol's solution and left on the slide for two to four minutes. It is important to avoid drying of the stain on the slide. If overstaining results dilution with water is possible. The excess iodine is then shaken off and the slide dried by pressure between two pieces of filter paper. No cover slip or immersion oil is needed. (2 references)

Lawrence L. Garner.

Smith, C. H., Gilkes, M. J. and Sowa, J. **A report on the attempted isolation of the virus of trachoma.** Brit. J. Ophth. 42:461-472, Aug., 1958.

The authors failed in their attempts to isolate the trachoma virus in HeLa cells, conjunctival cells, mice or chick embryos. They discuss some of the probable causes of failure. (29 references)

Lawrence L. Garner.

Tanaka, C. and Watanabe, H. **A study of the relationship between adenovirus type 7 and epidemic keratoconjunctivitis.** Acta Soc. Ophth. Japan 62:1255-1259, Aug., 1958.

Tanaka isolated adenovirus type 7 from a case of EKC with subepithelial punctate keratitis (AMA. Arch. Ophth. 59:48-54, 1958). The present study is undertaken to determine whether type 7 virus could cause the clinical picture of EKC in human volunteers. Tissue culture virus of this strain was inoculated into five volunteers and all developed an acute follicular conjunctivitis with preauricular adenopathy. The incubation period was short, two to four days. The first subject showed a slight epithelial keratitis; subepithelial keratitis did not appear in any eye. Adenovirus type 7 was re-isolated from one of the volunteers. A considerable rise in the neutralizing antibodies to the homologous virus was evident during the illness in four examined subjects. This experiment suggests that adenovirus type 7 can cause acute follicular conjunctivitis, but hardly typical EKC.

This article includes a discussion by Sugiura who inoculated the same virus into three human volunteers and observed a development of a "border line keratitis"

in one of the three. (1 figure, 1 table, 12 references) Yukihiko Mitsui.

Wakui, K. **Punctate keratitis in rabbits by inoculation with adenoviruses.** Acta Soc. Ophth. Japan **62**:1260-1290, Aug., 1958.

Wakui produced a subepithelial punctate keratitis in the rabbit cornea by subconjunctival injection of adenovirus type 8 and type 3. A single injection did not cause any change. Only after repeated injections was the keratitis brought about. The occurrence was 41 percent with type 8 and 10 percent with type 3. A similar keratitis was brought about by repeated subconjunctival injections of bovine serum; the occurrence was 40 percent. The culture fluid of HeLa cells, bovine vaccine and saline did not cause such a keratitis by repeated subconjunctival injections. On the basis of his data the author discusses the nature of the keratitis that is a manifestation of epidemic keratoconjunctivitis and thinks that it is an allergic manifestation in the cornea due to a viral affection of the conjunctiva. (5 figures, 5 tables, 35 references)

Yukihiko Mitsui.

3

VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Dreyer, Viggo. **Functional cone units.** Acta ophth. **36**:514-521, 1958.

The cones, particularly those in the fovea, are classically regarded as independent elements which convey a light-induced impulse to the visual centers via individual nerve paths. There is anatomical evidence that the rods are collected in groups; in the case of cones, however, we only know that statistically there ought to be some grouping, too, because the minimum separabile and the cone diameter do not coincide as has been assumed. In an ingenious experimental set-up, which must be studied in the original paper, this question was studied. These studies lead to the following conclusions. The cones in the fovea are able to cooperate in functional units. The number of cones in each group is maximally about 35, and such individual groups function independently of each other. The size of the group may vary with their state of adaptation. (6 figures, 8 references)

John J. Stern.

Ericson, Lennart A. **Daily variations in the inflow of the aqueous humour.** Acta ophth. **36**:381-385, 1958.

Using Rosengren's suction cup method, the author showed that the diurnal influx of aqueous is constant in persons working during the day and drops during sleep to about one-fourth of the day-time values. (2 figures, 9 references) John J. Stern.

Forsius, Henrik. **Analysis of vitreous and aqueous humour of enucleated eyes by paper electrophoresis.** Acta ophth. **36**: 569-579, 1958.

Forty-four eyes, enucleated for trauma, corneal ulcer, chronic uveitis, melanoma or absolute glaucoma, were used. The aqueous and vitreous were examined by paper electrophoresis and compared with serum in traumatic uveitis due to perforation of the cornea or of both the cornea and the sclera. In the presence of red and white blood cells the gamma and beta-globulin values were higher. In hyphema or vitreous hemorrhage similar proteinograms were obtained as with paper electrophoresis in which the beta-globulin predominated. The $alpha_2$-proteins were often of the same magnitude as in serum. The protein content of the vitreous was 4.74 percent in traumatic uveitis, 0.94 percent in absolute glaucoma, and 0.44 percent in corneal ulcer. (2 graphs, 5 tables, 26 references)

John J. Stern.

Gligorijevic, J., Jovanovic, M., Djurdjevic, D. and Petrovic, B. **The influence of ultrasound on the permeability of the cornea as determined by radioactive iodine.** Ann. d'ocul. **191**:363-371, May, 1958.

The authors measured the penetration of radioactive iodine into the cornea and aqueous from a solution of potassium iodide. An ultrasonic generator, frequency 800 Kc/sec., was used to drive the iodide into the eye. With weak applications the radioactive iodide tended to accumulate in the cornea. At higher intensities the iodide penetrated into the aqueous but the rate of disappearance was also increased apparently because of an increase in the permeability of the surrounding blood vessels. (4 figures)

David Shoch.

Gloster, J. and Greaves, D. P. **The effect of cervical sympathotomy on an ocular response to hypothalamic stimulation.** Brit. J. Ophth. **42**:385-393, July, 1958.

In laboratory cats, the hypothalmus was exposed and stimulated by electrical impulse; after the responses had been recorded, preganglionic cervical sympathotomy was performed on the same side as the stimulation and then the electrical shocks were repeated. The recorded results indicated a fall in intraocular pressure in the ipsilateral eye of 1 to 3 cm. saline. This change was constant and was accompanied in most cases by a rise in systemic blood pressure, retraction of the nictitating membrane and pupillary dilatation. After the sympathotomy the change in general blood pressure was not affected but the reduction in ocular tension, the pupillary dilatation and the retraction of the nictitating membrane were all abolished. This modification of the response must therefore be the result of the sympathotomy and the ocular changes produced by the electrical stimulation must be mediated by way of the ipsilateral cervical sympathetic nerve. These responses are not confined to the eye since simultaneous constriction of the vessels of the ear was also observed. (4 figures, 1 table, 10 references) Morris Kaplan.

Gordon, D. M. **Methylprednisolone in ophthalmology.** Metabolism **7**:569-573, July, 1958.

Methylprednisolone was used orally in a variety of ophthalmic conditions seen in a period of 11 months. Twenty-nine patients were given various dosages for periods of various length. The author concludes that methylprednisolone is "approximately 125 percent as effective as prednisone or prednisolone, and accordingly requires only about 80 percent of the dosage of these compounds." Water retention was found to be the chief side effect of this drug but this was easily controlled with diuretics in most cases. (6 figures) W. S. Hagler.

Hoshizumi, K. **Relation between ocular tension and systemic blood pressure.** Acta Soc. Ophth. Japan **62**:508-515, 626-631, 739-774, May, June, and July, 1958.

Rabbits were used in this experimental study of the relation between ocular tension and blood pressure. When a sudden increase in blood pressure is brought about by ligation of the descending aorta, the ocular tension increases with the blood pressure for the first 30 to 60 seconds. A late decrease in ocular tension follows in half an hour, even if the ligation persists. When the cervical sympathetic ganglion is removed, the late decrease of ocular tension does not occur. When the oculomotor nerve is blocked, the initial increase in ocular tension does not occur. (3 figures, 4 tables, 53 references)

Yukihiko Mitsui.

Kaivonen, Matti. **Comparative tests on the bactericidal effect of certain antisep-**

tics and antibiotics. Acta ophth. **36**:546-555, 1958.

Antibiotics are not recommended for prevention of blennorrhea in the newborn because of the existence of resistant strains. Silver nitrate and silver acetate are equally effective but lose much of their effect in the presence of chlorides and proteins. Amisept (a quaternary ammonium compound) is a potent bactericide, effective in the presence of chlorides and protein and thus superior to silver nitrate. It spreads easily into the fornices and is only mildly irritating. It is stable in solution and can be used to cleanse the skin about the eyes. (3 tables, 14 references) John J. Stern.

Kurachi, Y., Saito, K. and Kozawa, N. **Metabolism in inner and outer layers of the retina.** Jap. J. Ophth. **2**:127-134, May, 1958.

The tissue metabolism of the retina in white rabbits was measured by the Warburg indirect method after ligating the central retinal artery and also after ligating the ciliary vessels. Very little change occurred in the esterification ability of thiamine after ligating the central retinal artery. There was a marked decrease on ligating the ciliary vessels, indicating that the thiamine demand is three to four times as great in the outer layers of the retina. (3 tables, 8 references)
Irwin E. Gaynon.

Lugossy, Gyula. **The influence of antigens on the fluoresceine permeability of the blood-aqueous barrier.** Klin. Monatsbl. f. Augenh. **132**:848-855, 1958.

A prism-shaped fluorometer (a glass container filled with the dye) is attached to the slitlamp. The fluoresceine concentration in the anterior chamber corresponds to a certain thickness of the prism. The rate of diffusion of the dye can so be accurately measured. In patients with acute uveitis the rate of diffusion increases when a certain antigen, derived from extracts of prostate, tonsil, dental granuloma, or tuberculin, is given subcutaneously. (7 figures, 1 table, 9 references) Frederick C. Blodi.

Moi, Ivar. **Experiments on aqueous outflow in human and rabbit eyes.** Acta ophth. **36**:387-393, 1958.

Perfusion experiments in human and rabbit eyes, in vivo and in vitro, with increasing pressures up to 70 mm. Hg showed that the facility of outflow in in vitro experiments was slightly higher than in the corresponding in vivo experiments. This is due to a cessation of secretion of aqueous in vitro. Miotics and mydriatics had no effect on the facility of outflow in normal human and rabbit eyes. Four human eyes with absolute glaucoma showed little or no outflow; this indicates an organic stenosis of the outflow channels. Increased pressure in rabbit eyes caused increased outflow after 15 to 25 minutes. In a hypotonic eye with retinal detachment, a greatly increased facility of outflow was found. In an experiment in a patient in whom the outflow facility had previously been normal there was a great increase of the facility of outflow after luxation of the lens into the vitreous. (3 figures, 1 table, 14 references) John J. Stern.

Nishiyama, S. **Toxicity of some medicaments on the conjunctival epithelial cells: a study by tissue culture technique.** Acta Soc. Ophth. Japan **62**:846-868, July, 1958.

The toxicity of some medicaments for conjunctival epithelial cells was examined in vitro. A cytopathogenic effect of the medicaments in causing degeneration and detachment of the cells is studied by a tissue culture technique. Chang's strain of human conjunctival epithelium was used. The minimum inhibitory concentration in log of mg/ml of examined medicaments are: tetracycline −2, streptomycin −2, penicillin −1, sarcomycin −1, polymyxin

−1, $ZnCO_4$ 0, pilocarpine 0, atropine −1, cocaine 0, and $HgO \cdot Hg(CN)_2$ −3. The toxic effect of the same medicaments on the HeLa cells is also examined as a control. The epithelial cells of the conjunctiva show ten to one hundred times greater resistance against $ZnSO_4$ than HeLa cells, while the former show a lower resistance than the latter against most of the antibiotics. (15 figures, 21 tables, 12 references) Yukihiko Mitsui.

Senoo, K. **Biochemical studies of heterokeratoplasty.** Acta Soc. Ophth. Japan **62**: 663-670, 876-880, June, July, 1958.

The author reports a basic experiment in heterokeratoplasty. First, the amino acids of the cornea, aqueous and serum were measured by paper chromatography in various animals. Then chick cornea was transplanted into rabbit cornea. A good result was obtained in 13 trials and a poor result in seven. In the former, the amino acid fractions of the donor cornea changed gradually in 40 days to reach a similar composition to that of the receptor cornea. The author concludes that in heterokeratoplasty it is advisable to immerse the donor cornea in the serum of the receptor animals for a certain period of time before transplanting it into the receptor eye. (8 figures, 8 tables, 10 references)

Yukihiko Mitsui.

Seto, Y. **Influence of carbon disulfide on the ERG in rabbits.** Acta Soc. Ophth. Japan **62**:951-961, Aug., 1958.

After a slight exposure to CS_2 the b-potential of the ERG in rabbits decreases but the c-potential does so only after an intensive exposure. It is therefore supposed that a slight intoxication affects the synapse in the retina, and an intensive intoxication the receptor itself. (8 figures, 3 tables, 40 references)

Yukihiko Mitsui.

Tsuchiya, H. **Influence of fever therapy on the caliber of the retinal blood vessels.** Acta Soc. Ophth. Japan **62**:1032-1061, Aug., 1958.

In fever therapy the systemic blood pressure shows an initial rise at the beginning of the temperature rise; it is followed by a secondary fall corresponding to the maintenance of high fever. The retinal vessels show an initial contraction and a secondary dilatation. (19 figures, 23 tables, 36 references) Yukihiko Mitsui.

Yamane, T. **Influence of ACTH on the ERG in rabbits.** Acta Soc. Ophth. Japan **62**:938-950, Aug., 1958.

The author studied the influence of ACTH on the ERG in rabbits. When 1.0 to 10 mg. of ACTH is given, the amplitude of the b-potential begins to increase in five minutes and reaches the maximum in one hour. A similar but a greater increase occurs in the c-potential also. When ACTH is given every day for a few weeks, the b-potential begins to react poorly while the c-potential does not. On the basis of these facts Yamane discusses the mode of action of ACTH on the ERG. (7 figures, 5 tables, 102 references)

Yukihiko Mitsui.

4

PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Gardiner, P. A. **Dietary treatment of myopia in children.** Lancet 1:1152-1155, May 31, 1958.

Myopic children grow more irregularly and mature earlier. The rate of myopic change coincides both in time and degree with spurts of physical growth. Increase in myopia is greater and more common in children whose diet is deficient in animal protein.

An attempt was made to give the treated children (91) ten percent of their caloric intake as animal protein. In the

younger children the deterioration was greater in the controls by one half diopter per year. The children who had the most protein deteriorated the least, and in those over 12 years of age there was actual improvement. (1 figure, 5 tables, 6 references) Irwin E. Gaynon.

Gramberg-Danielsen, B. **Localization and causes of optical delusions concerning the size.** Klin. Monatsbl. f. Augenh. **132**: 647-655, 1958.

A central dysmegalopsia may occur in young or psychotic patients or in patients with Graves' disease. Peripheral types of this condition may be produced by ocular defects (anomalies of the refractive media, macular edema, chorioretinitis, macular detachment), damage to the visual pathways (inflammations, tumors, vascular disturbances), or by disturbances of convergence and accommodation. (3 figures, 26 references) Frederick C. Blodi.

Hager, G. **The field of gaze and of panoramic vision in ocular and systemic diseases.** Klin. Monatsbl. f. Augenh. **132**:790-796, 1958.

The field of gaze is decreased in all types of ophthalmoplegia. The field of panoramic vision, in which head, shoulder and pelvis can be moved, will decrease especially with affections of the joints, for example in Marie-Strumpell's disease. (14 figures, 24 references)

Frederick C. Blodi.

Monjé, Manfred. **The sensitivity for colored targets in the visual field of color-blind eyes.** Klin. Monatsbl. f. Augenh. **132**:635-646, 1958.

The threshold values for colored lights were determined at a certain level of light adaptation for various retinal areas. These absolute values are not influenced by the wearing of colored glasses prior to the test, nor by a color blindness of the eyes which are examined, except for protanopes and protanomales. Here the sensitivity for longer waves decreases. In this way protanopes can be differentiated from deuteranopes. In the first type the white substance is also affected. The latter must be independent from the color sense and is not identical with visual purple. (5 figures, 1 table, 37 references)

Frederick C. Blodi.

Pugh, Mary. **Visual distortion in amblyopia.** Brit. J. Ophth. **42**:449-460, Aug., 1958.

The visual acuity of an amblyopic eye appears to be one, two, or three grades worse when tested on a whole line of a Snellen chart than when these letters are isolated, or separate Snellen letters are shown. The difficulty a patient has in reading consecutive letters in a row appears to be due to an effect of one letter overlapping another and the patient's inability to disentangle one letter from another. The power to resolve single letters varies with the shape of the letter. This was found to be due to a distortion of outline which occurred when the patient fixed one side of the letter but was not present when he fixed the other side. When the area of pattern disturbance coincide with a special feature of the letter's shape, that letter is not resolved correctly. The direction of pattern disturbance is consistent and constant and appears to be directly related to the heterotropia which is present binocularly. This direction appears to be opposite to that of the heterotropia, but may be modified by a significant astigmatic error acting as a secondary factor.

This directional effect can be corrected with treatment, thus proving that it is not due to irregular astigmatism or other organic irreversible conditions. The effect is quite unlike that due to suppression. The graduated single letters used subtend 10 to 7.5 minutes of arc at the fovea and therefore must be resolved by the slender

cones on the floor of the fovea. There seems to be no accepted theory of vision to account for these effects, but if the constantly tilted position of the visual axis causes a tilting of the foveal receptors, an explanation can be suggested. The possibility of phototropic movements may possibly be considered. (3 figures, 1 table, 20 references) Author's summary.

Suzuki, T., Mori, H., Yoshino, T., Shiwa, K. and Murase, T. **Electroretinogram by congenital total color anomaly with optic nerve lesion.** Acta Soc. Ophth. Japan Aug., 1958.

A case of congenital, total color anomaly is reported. One eye is completely blind as a result of traumatic optic nerve contusion. The ERG of the uninjured eye has a considerably lower b- and c-potential in comparison with that of normal individuals. However, the ERG of the blind eye is much the same as that of normal persons. It is interesting to know that by a blocking of the optic nerve, the low potential of the ERG by total color anomaly has disappeared. The authors consider that the low potential of the ERG in total color anomaly may be due to an interference from the cortical center through the optic nerve. (5 figures, 3 tables, 24 references) Yukihiko Mitsui.

5

DIAGNOSIS AND THERAPY

Alfano, Joseph E. **Rubber-tipped metal applicator.** Tr. Am. Acad. Ophth. **62**:137, Jan.-Feb., 1958.

To avoid introduction of cotton fibrils when retracting a limbal-based flap in cataract or glaucoma surgery, the author advises a metal applicator with a rubber tip. (1 figure) Harry Horwich.

Amanuma, H. **Capillary fragility of the episclera.** Acta Soc. Ophth. Japan **62**:533-542, 686-692, May, June, 1958.

The fragility of the episcleral capillaries is measured by counting petechiae after exposure to negative pressure. The fragility increases with retinal angiosclerosis. The fragility of the skin capillaries does not give an indication of retinal angiosclerosis. The fragility of skin capillaries can be improved by some medicaments but not that of the scleral capillaries. (8 figures, 15 tables, 46 references) Yukihiko Mitsui.

Braga, P. and Renata, E. **Critical study of the ophthalmoscopic classifications of arterial hypertension.** Rev. brasil. oftal. **17**:189-212, June, 1958.

The author discusses briefly all different theories which have been made the basis for a classification of the ophthalmoscopic signs in hypertension. (24 references) Walter Mayer.

Cardell, J. D. M. **Refraction in marked ptosis and blepharochalasis.** Brit. J. Ophth. **42**:441, July, 1958.

In cases of marked interference, refraction can be facilitated by lifting the lid with a wire fitted into the back cell of the trial frame. The wire must be so arranged that it does not press on the globe and produce an artificial astigmatism. (1 figure) Morris Kaplan.

Cardoso Rebocho, P. R., Guimarães, W., deBarros Mattos, M. and de Azevedo, M. L. **Clinical significance of ophthalmoscopy in arterial hypertension.** Rev. brasil. oftal. **17**:235-246, June, 1958.

The authors review the general findings in hypertension and correlate them with the ophthalmoscopic findings. They feel that the ophthalmoscopic examination is important in the classification of degree of the disease and also in evaluating the effect of some of the newer drugs used in the treatment of hypertension. (1 table, 22 references) Walter Mayer.

Edmund, Jens. **The value of the eosinophile count in traumatic lesions of the eye.** Acta ophth. **36**:455-467, 1958.

A relationship appears to exist between sympathetic ophthalmia and eosinophilia. Enucleation of the injured eye becomes imperative only if and when the eosinophile count rises. (8 figures, 1 table)

John J. Stern.

Ellis, Philip P. **The dangers of ophthalmic corticosteroids.** Iowa St. Med. Soc. J. **48**:355-357, July, 1958.

Local corticosteroids are contraindicated in viral and fungal infections of the cornea, and should be avoided in corneal ulcers due to bacteria unless the proper antibiotic is used at the same time. It can be dangerous in certain forms of granulomatous iritis associated with syphilis, tuberculosis and toxoplasmosis.

"The indiscriminate use of corticosteroids, either alone or in combination with antibiotics, may lead to a decrease in resistance and reparative processes, may spread the infection, and in some instances may lead to the actual loss of the eyeball." (4 references)

Irwin E. Gaynon.

Gordon, Dan M. **Hand fundus camera.** Tr. Am. Acad. Ophth. **62**:134, Jan.-Feb., 1958.

A Japanese ophthalmoscope carrying a monoflex camera and powered by a Xenon electronic flashlamp is described by photograph. (1 figure) Harry Horwich.

Hollenhorst, R. W. **Ophthalmodynamometry and intracranial vascular disease.** Med. Cl. North America pp. 951-958, July, 1958.

The principles involved and the technique used in ophthalmodynamometry and its significance in various systemic conditions are discussed. In a large series of cases it has been found that a difference of 8 to 10 mm. systolic or diastolic pressure in the retinal arteries in the pair of eyes is indicative of diminished blood flow in the carotid artery on the side with the lower pressure. Among patients with intracranial vascular disease this is often a valuable aid in diagnosis. Some 70 percent of patients with thrombosis or intermittent insufficiency of the carotid artery will have a significantly lower pressure in the retinal artery of the affected side. Approximately 56 percent of the patients with carotid artery insufficiency will have attacks of visual loss on the affected side whereas only occasionally will this occur in those with a thrombosis of the internal carotid artery.

Many patients who have sustained an occlusion of the basilar or vertebral arteries will have greatly increased retinal artery pressure bilaterally. Hypertensives often have an elevated retinal artery pressure which is frequently disproportionately high in comparison to the brachial blood pressure. Retinal artery pressures are also elevated in the presence of increased intracranial pressure, but only prior to the appearance of papilledema. The determination of the retinal artery pressure has also been found valuable in the diagnosis of postural hypotension. (19 references) W. S. Hagler.

Kato, T., Toyama, S., Furusawa, T. and Mita, S. **CRP-test in the diagnosis of Behçet's syndrome.** Acta Soc. Ophth. Japan **62**:800-805, July, 1958.

The result of the C-reactive protein test (J. Exp. Med. 52:561, 1930) in 95 patients is reported. This reaction is highly positive in cases of endogenous uveitis. The reaction is particularly evident in Behçet's syndrome, and is positive throughout the course of the disease. It is, therefore, valuable in the early diagnosis of this condition. (5 tables, 12 references)

Yukihiko Mitsui.

King, J. H. Jr., Chavan, S. B. and Furness, C. W. **An instrument for lamellar keratoplasty.** Tr. Am. Acad. Ophth. **62**: 132-133, Jan.-Feb., 1958.

An instrument like a chalazion clamp is described. The lower solid blade has a convexity of curvature matching the cornea, and the upper ring blade comes in 8 and 11 mm. sizes. The excised donor cornea is placed between the two blades and held secured for trephination and dissection. (1 figure) Harry Horwich.

Krug, A. and Lobo, F. **The pathologic significance of the ophthalmoscopic signs in arterial hypertension.** Rev. brasil. oftal. 17:215-220, June, 1958.

The authors discuss the pathologic findings which are the basis of increased arterial reflexes, vascular sheating, arterio-venous crossing defects, irregularities in the vascular diameter, tortuosity of vessels, aneurysms, generalized constriction of the retinal arterioles, retinal edema, hard and cotton wool exudates, macular star and deep and superficial hemorrhages and vascular thrombosis. (22 references) Walter Mayer.

Krwawicz, T. and Zagorski, K. **Experimental electrochemical removal of copper splinters from the isolated vitreous.** Brit. J. Ophth. 42:494-500, Aug., 1958.

The authors used bovine vitreous as an electrolite solution and sprinkled small copper splinters in it. They placed a small electrode (anode) near the copper and a fine platinum needle as an inactive electrode (cathode). A small electric dry cell was used as the source of current. It was shown that copper splinters can be dissolved; the size of the splinter and the presence of insulation on the electrodes are important factors. On the basis of this experiment 79 percent of the copper particles placed in bovine vitreous could be precipitated electrochemically. (2 figures, 2 tables, 7 references)

Lawrence L. Garner.

Leydhecker, Wolfgang. **The range of error in using a standardized tonometer on the living human eye.** Klin. Monatsbl. f. Augenh. 132:855-860, 1958.

In 2,285 eyes the tension was determined with a Schiøtz tonometer. Two measurements were done in a short interval. The interval was long enough to allow the patient to move or adjust his head. In only five eyes was there a difference of more than one scale reading between the two measurements. In 99.8 percent of all eyes the difference between the two measurements was less than one scale reading. In 75 percent of the eyes the measurements were identical. (1 table, 4 references) Frederick C. Blodi.

Mathieu, Peter L. **Comparison study: silver nitrate and oxytetracycline in newborn eyes.** A.M.A. J. Dis. Child. 95:609-611, June, 1958.

Oxytetracycline administered topically is an effective prophylactic measure, does not irritate the eye, and rarely causes sensitization. Of 1,139 newborn infants observed during the first five days of life, six percent of those treated with oxytetracycline had conjunctival discharge, compared with 46 percent of those given silver nitrate. (1 figure, 1 table, 10 references)

Irwin E. Gaynon.

McOwan, B. M. **Two cases from Malaya.** Brit. J. Ophth. 42:437, July, 1958.

Dramatic pictures of a case of strabismus fixus and one of tremendous orbital tumor are presented. (4 figures)

Morris Kaplan.

Mishima, S. **Biomicroscopy of human eyes with polarized light.** Acta Soc. Ophth. Japan 62:380-384, 492-497, April. May, 1958.

In slitlamp microscopy of the cornea with polarized light the so-called "dark cross" is actually a "dark hyperbola", and it is a representative of the deep-layer structure of the cornea. The colored band. which seems to be on the iris is actually due to double refraction of the rays in the

cornea. The micelle structure of the corneal fibers is clearly seen by a polarized-light biomicroscopy. (10 figures, 15 references) Yukihiko Mitsui.

Niedermeier, Siegfried. **Spectrophotographic measurements of the choroid.** Klin. Monatsbl. f. Augenh. **132**:828-839, 1958.

For this method a Sachs light with special filter is used. It is held against the sclera. The spectroscope is over the center of the pupil. The diameter of the pupil is compensated for by the duration of exposure. The average thickness of the choroid 16 mm. behind the limbus was found to be 0.23 mm. The values were much higher in glaucomatous eyes. The drop of intraocular pressure after a retrobulbar procaine injection is apparently due to a decreased choroidal volume. (7 figures, 1 table, 15 references) Frederick C. Blodi.

Odquist, Bertil. **New permanent magnets for ophthalmological use.** Acta Ophth. **36**:471, 1958.

A permanent magnet manufactured by KIFA in Stockholm is described. It is as powerful as modern electro-magnets and has obvious advantages. John J. Stern.

Potts, A. M. and Brown, M. C. **A color television ophthalmoscope.** Tr. Am. Acad. Ophth. **62**:136-137, Jan.-Feb., 1958.

The many actual and theoretical advantages of being able to project a color television image of the fundus are enumerated. Illumination is by a May prism system, or by zirconium arc through the objective lens of the ophthalmoscope. The objective of a fundus camera directs the rays through a field-sequential (CBS) color camera chain. (1 figure) Harry Horwich.

Rasmussen, K. and Schlegel, C. **Primary operation in zygomatico-maxillary fractures with diplopia.** Acta ophth. **36**: 468-470, 1958.

Zygomatico-maxillary fractures with diplopia call for early reposition and fixation of the fragments. (1 table) John J. Stern.

Rezende, C. **Present concept of arterial hypertension and its eye manifestations.** Rev. brasil. oftal. **17**:151-170, June, 1958.

The author gives a historical summary of the findings in arterial hypertension, mentioning briefly the most important concepts about this disease since the time of Bright. A large portion of this paper is devoted to the ophthalmoscopic signs found in arterial hypertension and to the classification of Wagener and Keith. The author concludes his paper with a fairly detailed discussion of the pathogenesis of all the various retinal lesions which can be found in hypertensive and arteriosclerotic retinopathies. (2 figures, 32 references) Walter Mayer.

Stein, Lester. **Combined white and black light fluorescent lamp examining unit.** Tr. Am. Acad. Ophth. **62**:131-132, Jan.-Feb., 1958.

A means of mounting a pair of white light fluorescent tubes and a pair of Wood's light tubes in the same Burton unit with a Gaynor switch set for rapid alternation is described. (2 figures) Harry Horwich.

Stein, Lester. **Floor stand mounting for high intensity Wood's light.** Tr. Am. Acad. Ophth. **62**:129, Jan.-Feb., 1958.

The Luxo adjustable bracket is described as adapted for a safe, convenient carrier for the hot mercury-arc Wood's lamp. (1 figure, 1 reference) Harry Horwich.

Stein, Lester. **New circline fluorescent tube photokeratograph.** Tr. Am. Acad. Ophth. **62**:130, Jan.-Feb., 1958.

An instrument for photographically recording Placido's multicircular corneal illuminator is described. It consists of a

camera suitably mounted in the center of three concentric fluorescent circular light fixtures. (2 figures, 1 reference)

Harry Horwich.

Toledo, P. and Fonseca, J. **Ophthalmoscopic signs in arterial hypertension.** Rev. brasil. oftal. 17:173-187, June, 1958.

The authors discuss the various ophthalmoscopic signs found in hypertensive retinopathy: decreased transparency of vessell wall, increase in the arteriolar light reflex, sheathing, arterio-venous crossings, irregularities in the diameter of the vessels, neovascularization, aneurysms, exudates, retinal edema, hemorrhage, papilledema, vascular occlusions, and changes in pigmentation in the macular area. (8 figures, 24 references)

Walter Mayer.

Treiger, R. and Cavalcante, A. **Ophthalmoscopic findings in the hypertensive disease of pregnancy.** Rev. brasil. oftal. 17: 223-233, June, 1958.

The authors review the literature on the ophthalmoscopic findings in hypertension encountered in pregnancy and, because of the numerous changes, they conclude that this examination should be done routinely for diagnostic, prognostic and therapeutic purposes. (1 table, 34 references) Walter Mayer.

Vannas, S. **A method for correction of the retracted upper eyelid.** Acta ophth. 36:444-454, 1957.

The retraction of the upper lid after enucleation is corrected by implantation of surgical sponge behind the tarso-orbital fascia. The results were good. (14 figures, 9 references) John J. Stern.

Weigelin, E. **The physical basis of ocular dynamometry.** Ann. d'ocul. 191:333-343, May, 1958.

The author feels that dynamometry has not received its rightful place in ophthalmology because of a general misunderstanding of the factors on which this diagnostic procedure is based. He shows that the pressure recorded by the dynamometer is that arterial pressure found along the course of the ophthalmic artery. It is lower than the pressure at the origin of the ophthalmic artery and higher than the pressure at the origin of the central retinal artery. Local vascular alterations in the eye do not affect the dynanometer readings but alterations in the carotid supply affect it greatly. By virtue of this finding dynanometry is useful in the following conditions: obliterating thromboangeitis of the common or internal carotid or of its larger branches, cerebro-vascular sclerosis, arterial hypertension, and cephalalgias of vascular origin. (4 figures, 12 references) David Shoch.

Wendland, John P. **Small curved probe for Berman locator.** Tr. Am. Acad. Ophth. 62:135, Jan.-Feb., 1958.

A curved probe has now been developed to facilitate locating magnetic intraocular foreign bodies posterior to the equator. It will detect such bodies from a distance approximately six times their diameter. The sensitivity is somewhat less than that of the old straight probe. (1 figure)

Harry Horwich.

Young, J. Horton. **Ocular cases of medical interest.** Brit. J. Ophth. 42:438-440, July, 1958.

The author describes four cases of ocular disease seen during several years of military service: myopathic atrophy, caterpillar hairs in the eye, accidental solanaceous mydriasis and melanosarcoma of the macular area. The mydriasis occurred in a soldier who accidently received the juice of a Mediterranean fruit in both eyes. The juice was found to contain hyocyamine equivalent to 2 percent solution of atropine. Morris Kaplan.

6
OCULAR MOTILITY

Berke, Raynold N. **Requisites for postoperative third degree fusion.** Tr. Am. Acad. Ophth. 62:38-53, Jan.-Feb., 1958.

The author has analyzed a series of 256 consecutive surgical strabismus patients. The series did not include patients with organic sensory defects or acquired muscle pareses. Factors which were found to be of little or equivocal importance in the development postoperatively of third degree fusion are: amount of deviation (in any direction), refractive error, type of operation done, age at operation, orthoptics, position of the eyes under general anesthesia, anatomic abnormalities and postoperative adhesions, heredity, anomalous retinal correspondence, and manifest lesions of the optomotor pathways. The following factors may militate against success: the squint dating from birth, long duration as related to the age of the patient, amblyopia, a residual horizontal tropia of over 10 prism diopters or a vertical one over 2 prism diopters in the primary position. Intermittency, of course, greatly improves the prognosis. (2 figures, 10 tables, 30 references)

Harry Horwich.

Ehrich, Wulf. **Binocular vision after operations for hypertropia.** Klin. Monatsbl. f. Augenh. 132:681-690, 1958.

Ehrich evaluated 56 patients; the quality of binocular vision improved in 42 after the surgery. Nearly half of the patients had binocular vision before the operation. The results are better when the patient is young and when there is no appreciable horizontal deviation. (6 tables, 33 references)

Frederick C. Blodi.

Frandsen, D. **Some results from a clinical-statistical survey on strabismus among Copenhagen children.** Acta ophth. 36:488-498, 1958.

Six percent of the Copenhagen children are premature. Among all squinting children, 11.5 percent were premature. Left-handedness occurs in 12 percent of squinting children and in 6 percent of the normal population. It seems that left-handedness and prematurity are in some way correlated with strabismus. (11 figures)

John J. Stern.

Haensel, W. **Experiences with the myectomy of the inferior oblique muscle.** Klin. Monatsbl. f. Augenh. 132:879-883, 1958.

A vertical component is frequent in horizontal strabismus. Among 450 operations for strabismus a myectomy of the inferior oblique muscle of one or both eyes had to be done in 160 cases. A recession of the muscle is thought to be too difficult an operation. (22 references)

Frederick C. Blodi.

Hager, G. **The field of gaze (with fixed and moving head) in normal persons.** Klin. Monatsbl. f. Augenh. 132:656-670, 1958.

Examinations of 348 normal persons were done on the gaze perimeter of Comberg. There was a decrease of the field with age in all meridians. The field of gaze can also be determined when only the stance of the legs remains fixed. The patient can move not only his eyes but also head, neck, shoulder and pelvis. This field also decreases with age. (7 charts, 15 figures, 7 tables, 1 reference)

Frederick C. Blodi.

Hamburger, F. A. **The application of vasodilators and vasoconstrictors to the external eye.** Klin. Monatsbl. f. Augenh. 132:735-740, 1958.

Subconjunctival injections of Priscoline are of great value in the treatment of chemical burns of the eye. Priscoline can also be used locally in ointment or solution. The drug is also of value in the treatment of bullous keratitis.

Frederick C. Blodi.

Herberg, H. J. and Schilf, E. **Neurologic considerations in a case of voluntary pendulating nystagmus.** Klin. Monatsbl. f. Augenh. **132**:780-790, 1958.

A 49-year-old man was observed who could voluntarily produce a pendulating nystagmus. The movements were quite fast as could be recorded by electro-oculography. Such high frequency resembles a tremor of other skeletal muscles. (3 figures, 24 references) Frederick C. Blodi.

Mackensen, G. **The theory of optokinetic nystagmus.** Klin. Monatsbl. f. Augenh. **132**:769-780, 1958.

A number of observations are cited which speak against the classical (Barany) theory of the etiology of optokinetic nystagmus which associates the fixating eyes closely with the objects of a moving environment. One observation was made on a patient with unilateral ophthalmoplegia. When a moving object was presented to this eye, the sound, but covered eye, showed optokinetic nystagmus. Another factor is the after-nystagmus which follows the optokinetic nystagmus. The existence of this after-nystagmus was heretofore denied by some authors. MacKensen could convincingly demonstrate it by electro-oculography. The duration of the stimulus can be shortened to 0.05 sec. and still elicit a nystagmus. While the stimulus remains unchanged, the nystagmus increases during the first six minutes. In addition to the optical regulation a central influence must be assumed (6 figures, 31 references)

Frederick C. Blodi.

Mertens, H.-G., Erlo, and Papst, W. **The ocular myopathies: oligosymptomatic ocular myositis (pseudomyasthenia).** Nervenarzt **29**:213-226, May 20, 1958.

The authors present the third part of their investigations and discuss seven cases of a previously undescribed form of ocular myositis. The clinical appearance is that of ptosis and ocular muscle palsy similar to that of myasthenia gravis but prostigmine is ineffective. Electromyography indicates a myopathy and good improvement is obtained with cortisone. (3 tables, 8 figures, 30 references)

Edward U. Murphy.

Møller, Poul Martin. **The influence of anesthesia and premedication on the angle of squint.** Acta ophth. **36**:499-498, 1958.

In about 50 percent of cases the angle of convergence was considerably reduced after premedication with morphine and atropine, and also under ether anesthesia. Children whose angle of squint was not affected by premedication also showed no change during anesthesia. These preliminary findings call for an analysis and suggest that the surgeon should not be tempted to change his operative plan if the angle is altered by premedication or anesthesia. (5 references) John J. Stern.

Momosse, Hirofumi. **Studies of the action of the extraocular muscles in monocular movements by means of quantification of integrated EMG.** Jap. J. Ophth. **2**:108-122, May, 1958.

In horizontal movements of the eye, one of the horizontal rectus muscles contracts and the other is relaxed, while all of the vertical and oblique muscles maintain a constant level of discharge and never receive any increase in innervation. In vertical movements, two of the vertical rectus muscles contract, the other two are relaxed, and the electrical activity of the horizontal rectus muscles remains unchanged. In oblique movements, three muscles contract, and the remaining three are relaxed. (17 figures, 17 references)

Irwin E. Gaynon.

Nirankari, M. S. and Maudgal, M. C. **Incomitant strabismus of unusual origin.** Brit. J. Ophth. **42**:425-428, July, 1958.

A 16-year-old girl developed paresis of the left external rectus muscle after be-

ing stung by a wasp near the inner canthus. It was easily relieved by surgery. It is probable that the abduscens nerve endings were permanently paralysed by the wasp toxin.

A 25-year-old man noted aching in the outer aspect of one eye with gradual diplopia and esotropia. A nodular swelling was noted beneath the conjunctiva laterally and this was removed. It proved to be a cysticercus probably ingested in pork eaten one year before. Symptoms were entirely relieved. (2 figures, 1 reference) Morris Kaplan.

Papst, W., Esslen, E. and Mertens, H. G. **The ocular muscular dystrophy.** Klin. Monatsbl. f. Augenh. **132**:691-707, 1958.

The authors describe three patients with progressive external ophthalmoplegia. The electromyograms were consistent with a myopathy. Histologic examination of some extraocular and skeletal muscles revealed a marked dystrophy. (7 figures, 45 references) Frederick C. Blodi.

Piper, H. F. **Bilateral trochlearis paresis or Hertwig-Magendie strabismus.** Klin. Monatsbl. f. Augenh. **132**:671-681, 1958.

The first patient showed an alternating hypertropia of the adducted eye while the abducted eye fixates. During depression of the head to one side the ipsilateral eye moved upward. This condition followed a severe head injury and is interpreted as a variation of a Hertwig-Magendie strabismus. The other two patients had a bilateral trochlearis paresis. (3 figures, 29 references) Frederick C. Blodi.

7

CONJUNCTIVA, CORNEA, SCLERA

Aoki, Heihachi. **Acute trachoma in Japan.** Rev. intern. du trachome **35**:47-58, 1958.

The incidence rate of acute trachoma in Japan is low and its clinical symptoms and course are the same as in the trachoma produced by inoculation; however, the types of disease are different. The term paratrachoma should not be given to inclusion conjunctivitis with inclusion bodies, acute course and good prognosis, until the virus of trachoma and paratrachoma is isolated. (1 figure, 3 tables, 18 references) José A. Ferreira.

Eisum, E. F. **Experimental corrosions of the cornea.** Acta ophth. **36**:483-487, 1958.

Hydrochloric acid burns of the cornea were followed within one hour by liberating the conjunctiva from the cornea in one sector near the limbus. More vascularization of the cornea was observed in the sector thus treated. This is desirable for better circulation and tissue repair and it is recommended for patients with acid burns. (1 figure) John J. Stern.

Gilkes, M. J., Smith, C. H. and Sowa, J. **Trachoma inclusions and penicillin.** Brit. J. Ophth. **42**:478-482, Aug., 1958.

Five cases are described in which 900,000 units of procaine penicillin were given intramuscularly on each of five days. No other local or systemic therapy was given. The use of an iodine-staining method revealed degenerative changes in the inclusion bodies and their disappearance in cases that had been positive for a long time. Because the series is small and the period of observation minimal the authors do not present the dosage as curative of trachoma. (12 references) Lawrence L. Garner.

Harms, H. **Permanent covering of the cornea with a conjunctival flap.** Klin. Monatsbl. f. Augenh. **132**:707-714, 1958.

A large flap is prepared and inverted so that conjunctival epithelium rests on the corneal surface. At the limbus the flap is pushed under the mobilized con-

junctiva so that the raw surfaces of the flap and of the limbal conjunctiva will adhere to each other. The flap is now sutured to the adjacent conjunctiva by a running mattress suture which does not perforate conjunctival epithelium. Such covering is especially indicated in cases of lagophthalmus, corneal anesthesia, and mechanical irritation. The cornea is still visible under the flap. The flap can be removed in stages. (6 figures)

Frederick C. Blodi.

Hobbs, H. E. and Calnan, C. D. **The ocular complications of chloroquine therapy.** Lancet 1:1207-1209, June 7, 1958.

Corneal changes secondary to chloroquine therapy were confined to the epithelium and the stroma immediately adjacent to Bowman's membrane. In some cases the appearance was that of whitish to yellow tinted lines in the corneal epithelium, irregularly curved, and concentrated just below the pupil, and with white dots scattered over the surface of the cornea. The second variety consisted of faint white dots scattered over the cornea, aggregated into curved lines resembling the "lines of force of a magnetic field". Except for one case, visual acuity returned to normal upon cessation of treatment. (3 figures, 3 references)

Irwin E. Gaynon.

Milklos, A. and Masovszky, L. **Protection of the graft with condom in keratoplasty.** Brit. J. Ophth. 42:401-405, July, 1958.

Several procedures for protection of the graft in keratoplasty have been described but none are considered satisfactory; the authors describe an additional method which has served very well for ten years. Condom rubber is used and is cut in a round patch 2 mm. greater in diameter than the graft to be employed. Several puncture holes are made in it and after proper sterilization it is loosely sutured into the episclera before surgery. After the keratoplasty the sutures are secured. It is removed on the eighth day. (4 figures, 4 references) Morris Kaplan.

Miura, R. **Electron microscopic study of trachoma granule.** Acta Soc. Ophth. Japan 62:1226-1239, Aug., 1958.

This is a study of the trachomatous conjunctiva in section by electron microscopy. The general histology is described. There are also some illustrations of epithelial cells which contain indisputable viral inclusions. (22 figures, 32 references)

Yukihiko Mitsui.

Romanes, G. J. **Infection with Micrococcus tetragenus as a complication of penetrating keratoplasty.** Brit. J. Ophth. 42: 429-432, July, 1958.

A 27-year-old man with progressive conical cornea became intolerant of contact lenses and sought keratoplasty. The donor eye for the surgery was removed with aseptic precautions and was treated with chloramphenicol. Its 48 hour culture was negative. The 8-mm. full-thickness graft was successfully placed with direct suturing and overlay sutures over egg membrane. The first dressing on the seventh day revealed purulent infection of the graft as well as of the host cornea. The culture revealed Micrococcus tetragenus sensitive only to chloramphenicol. This drug was injected beneath the conjunctiva daily along with large oral doses; it was also instilled as drops at 5-minute intervals day and night. After ten days the eye began to respond very rapidly and the patient was finally discharged with a clear graft and with vision of 6/36 unaided. Six weeks later the eye was completely quiet and clear and gave vision of 6/9 with a new contact lens. (2 figures, 1 reference) Morris Kaplan.

Salleras, A. **Surgical insertion of plastic corneas.** Arch. oftal. Buenos Aires 33:105-107, April, 1958.

As the results obtained with keratoplasty in cases of Fuchs' dystrophy, corneal burns, and in aphakic patients are poor, the insertion of an acrylic lens, 7 mm. in diameter, into the round window created by a central corneal trephination is thought to be worth trying. The procedure was used in two cases of opaque and vascularized cornea, in one of which two transplants had previously been performed without success. In both the operation could be carried out without immediate complications. As yet the follow-up is very short—four days in one case and two in the other. (8 references)

A. Urrets-Zavalia, Jr.

Sédan, Jean. **Three pilots who had trachoma.** Rev. intern. du trachome **35**:59-63, 1958.

Three pilots who almost had to leave their jobs because of active trachoma were kept working by regular and constant local treatment with tannic acid. To keep trachoma in the cicatricial stage in a long course of treatment is advisable.

José A. Ferreira.

Sezer, Necdet. **Characteristics of trachomatous inflammation.** Rev. intern. du trachome **35**:16-26, 1958.

Two stages can be recognized in the inflammatory reaction in trachoma. In a first stage there are inclusion bodies and purulent leucocytic infiltrates. In the second stage mitotic division of cells is conspicuous and retinculoendothelial cells abound. (10 figures) José A. Ferreira.

Sivasubramaniam, P. **Mode of action of tarsorrhaphy in neuroparalytic keratitis.** Brit. J. Ophth. **42**:483-485, Aug., 1958.

The author suggests that exposed areas are affected in neuroparalytic keratitis because of a lack of support of the cornea. He points out that supportive bandaging of an injured joint discourages edema around it. Performing a tarsorrhaphy furnishes this support and offers an excellent procedure to save not only the cornea, but possibly the eye. The procedure is excellent particularly in recurrent erosions of the cornea, vesiculations in herpes zoster and keratoplasty. Thirteen cases are described briefly. (8 references)

Lawrence L. Garner.

Sjögren, Henrik. **Corneal transplantation with punch forceps.** Acta ophth. **36**: 394-404, 1958.

A punch forceps is described which produces a graft and a hole of the same size with perpendicular sides, and without fringes and irregularities of Descemet's membrane and the endothelium. The results in 18 cases of corneal transplantation with this instrument are described. A clear graft was obtained in 15 cases, in ten of which there was a total leucoma with vascularization, which is usually regarded as a poor risk. (10 figures, 11 references) John J. Stern.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

von Burstin, Götz. **A case of choroideremia with electroretinographic examination.** Klin. Monatsbl. f. Augenh. **132**:869-874, 1958.

Three patients, a father and two daughters, are described. The ERG was abolished in the father and normal in the two carriers. (3 figures, 14 references)

Frederick C. Blodi.

Hervouet, F. and François, P. **An angioma of the choroid.** Ann. d'ocul. **191**:344-357, May, 1958.

The authors describe microscopic preparations of an eye with an angioma of the choroid. It is of interest because the enucleation was performed before the development of increased tension and before any secondary changes had occurred. The

"pure" pathologic picture of angioma of the choroid is thus presented. (11 figures, 14 references) David Shoch.

Kato, Tadashi. **A clinical study on Behçet's syndrome. Occurrence rates of this syndrome in endogenous uveitis.** Jap. J. Ophth. **2**:122-127, May, 1958.

Of 38,053 outpatients, 1.35 percent or 512 patients had endogenous uveitis. Of these, 23.02 percent or 77 patients were considered to have a complete or incomplete mucocutaneous ocular syndrome. (6 tables, 11 references)

Irwin E. Gaynon.

Majima, S. **Serum protein, liver function and spinal fluid in Behçet's disease and idiopathic uveitis.** Acta Soc. Ophth. Japan **62**:1157-1175, Aug., 1958.

In Behçet's disease there is a definite decrease in serum albumin with an increase in serum globulin. In idiopathic uveitis there is a similar but a slighter change. In the former there is little disturbance in the liver function whereas in the latter there is a definite disturbance in the liver. In other types of uveitis there is no obvious change in the serum protein fractions. (5 figures, 17 tables, 77 references) Yukihiko Mitsui.

Ohrt, Vagn. **Rubeosis iridis diabetica.** Acta ophth. **36**:556-558, 1958.

The manner of formation of the new vessels in the iris is described in detail. It is assumed to be caused by a protracted venous congestion due to phlebopathy caused by the diabetes. There is no effective treatment. Cyclodiathermy seems to be the only procedure which brings about relief from the pain of secondary glaucoma and preservation of the amaurotic eye. John J. Stern.

Stein, R. **Replacement of traumatic iris prolapse.** Brit. J. Ophth. **42**:406-412, July, 1958.

The standard procedure for repair of a traumatic prolapse of the iris has always been pulling the iris out through the wound and excising it, leaving the iris, with the resultant coloboma, to fall back into its proper place. The dominant reason for not replacing the intact iris into the anterior chamber was the danger of introducing infection within the eye. However, with the proper use of antibiotics and steroids, this danger is practically eliminated and it is felt that the disadvantages of excising the prolapse are sufficient to discard the procedure. It is recommended that if the prolapse is not more than four days old and not more than 6 or 7 mm. in extent, it should be replaced intact. The procedure recommended is incision at the limbus with knife or keratome followed by the insertion of a cyclodialysis spatula and forcing the prolapse from within the chamber to return to its normal position. Air is injected and then the rupture of the cornea is sutured. This procedure has been used in 20 cases with uniformly good results and without any appreciable complications. (3 figures, 4 references) Morris Kaplan.

NEWS ITEMS

EDITED BY DONALD J. LYLE, M.D.
411 Oak Street, Cincinnati 19, Ohio

News items should reach the editor by the 10th of the month. For adequate publicity, notice of postgraduate courses and meetings should be received three months in advance.

DEATHS

Dr. Edwin F. Tait, Norristown, Pennsylvania, September 23, 1958.

Dr. Richard Parker Bell, Jr., Lakewood, Ohio, died July 26, 1958, aged 38 years.

Dr. Alfred Francis Luhr, Buffalo, New York, died August 4, 1958, aged 78 years.

Dr. William Ewing Shahan, Saint Louis, Missouri, died August 11, 1958, aged 81 years.

ANNOUNCEMENTS

ALL-INDIA MEETING

The All-India Ophthalmological Society will hold its annual meeting in Trivandrum on February 19, 20, and 21, 1959. The principal subject of this congress will be industrial ophthalmology. However, a number of free papers will also be included in the program. U. S. ophthalmologists interested in attending and addressing this meeting should contact:

Dr. Y. K. C. Pandit, Honorary General Secretary,
Bombay Mutual Building,
2nd Floor, Hornby Road,
Bombay 1, India

or

Dr. William John Holmes,
280 Alexander Young Building,
Honolulu 13, Hawaii.

UNITED KINGDOM ANNUAL CONGRESS

The annual congress of the Ophthalmological Society of the United Kingdom will be held at the Royal Society of Medicine, 1 Wimpole Street, London, W.1, on April 9, 10, and 11, 1959. The presidential address will be delivered by Mr. R. C. Davenport.

The subject for discussion will be "The swollen optic disc." This will be opened by Dr. S. P. Meadows and Mr. D. P. Greaves. Members who wish to take part in the subsequent discussion are advised to intimate their intention before the opening of the congress. It is emphasized that no member may speak for more than 10 minutes.

There will also be a short symposium on "The treatment of ocular injuries." The speakers will include Mr. M. J. Roper-Hall and Mr. John Whitwell.

The 1959 Bowman Lecture will be delivered by Prof. Louis Paufique of Lyons.

The annual dinner will be held at the Trocadero Restaurant on Thursday, April 9th. A Trade Exhibition will be held in the Cowdray Hall (next door to the Royal Society of Medicine).

On account of the difficulty in obtaining hotel accommodations in London, all members who will require it are advised to make their arrangements in good time.

Information may be obtained by writing:
Secretary, Ophthalmological Society
47 Lincoln's Inn Fields
London W.C. 2, England

FLORIDA SEMINAR

The Florida Midwinter Seminar of Ophthalmology and Otolaryngology will be presented in co-operation with the College of Medicine of the University of Florida and the University of Miami School of Medicine February 16 through 21, 1959, at the new Americana, 9701 Collins Avenue, Miami Beach. Outstanding speakers who will lecture on otolaryngology are John J. Conley, New York; George T. Harrell, Gainesville, Florida; William C. Huffman, Iowa City, Iowa; P. E. Ireland, Toronto, Canada; and Arthur L. Juers, Louisville, Kentucky.

The lectures on ophthalmology will be presented on February 19th, 20th, and 21st. The lecturers for these courses will be Frank D. Carroll, New York; Paul A. Chandler, Boston; John W. Henderson, Ann Arbor, Michigan; and Harvey E. Thorpe, Pittsburgh.

The registration fee for the seminar is $50.00. A check for $10.00 payable to the Florida Midwinter Seminar must accompany application. This is not returnable. The remainder of the registration fee will be paid at the seminar desk at the Americana Hotel on arrival. All the facilities of the Americana, including the beach and swimming pool, are available to all the registrants of the seminar and their families. For further information or registration write:

Dr. Kenneth Whitmer
550 Brickell Avenue
Miami, Florida

RESEARCH STUDY CLUB

On the ophthalmology program for the 28th annual midwinter convention of the Research Study Club of Los Angeles, to be held at the Ambassador Hotel, Los Angeles, January 26 through January 30, 1959, are: Prof. A. Franceschetti, Geneva, Switzerland; Dr. Arthur G. DeVoe, New York; Dr. Lester T. Jones, Portland, Oregon; Dr. Arthur

Jampolsky, San Francisco; and Dr. Carrol L. Weeks, Los Angeles. This year, during the meeting, for an additional fee of $50.00, a special course on cardiac resuscitation will be given by Dr. William P. Mikkelsen. In order to be eligible for attendance at the convention, all applicants must be members in good standing of the American Medical Association. The fee for the course, $110.00, includes the cost of all round-table luncheons. For application or further information write:

Dr. Norman Jesberg, treasurer
500 South Lucas Avenue
Los Angeles 17, California

Gregg prize

The Ophthalmological Society of New South Wales announces that the Norman McAlister Gregg Prize will be awarded in 1959. The closing date for entries will be October 31, 1959, at midday.

The prize of 100 guineas (Aust.) and a bronze medallion will be awarded to the British subject whose submitted original work, on any subject, is deemed to be the most valuable contribution to knowledge in ophthalmology.

Intending candidates should notify the secretary of the society, Dr. John Hornbrook, 147 Macquarie Street, Sydney, at least two months before the closing date. Regulations for the prize (a copy of which follows) may be obtained from the secretary.

Regulations 1958-59, Norman McAlister Gregg Prize.

1. Subject to the rules, the prize shall be awarded to the candidate whose work is deemed to be the most valuable contribution to knowledge in ophthalmology. In the event of the council being of opinion that no work is of sufficient merit it shall not award the prize.

2. A candidate must be, and declare that he is, a British subject.

3. The work submitted must be in the English language. It must be the candidate's own original work and written since the prize was last awarded (August, 1955). If such work has previously been submitted for publication, details must be supplied by the candidate.

4. The council shall appoint examiners for each competition and the council's decision as to the winning entry shall be final and conclusive.

5. Intending candidates shall notify the secretary of the society of their intention to submit contributions at least two months before the closing date.

6. Three copies of the work are to be submitted. It is to be typewritten and double spaced. Each work shall bear a motto and shall be accompanied by a sealed envelope containing the author's name and qualifications.

7. Entries will close for the present prize on October 31, 1959.

Basic science lectures

The Department of Ophthalmology, Washington Hospital Center, 110 Irving Street, N.W., Washington 10, D.C., is offering a series of lectures on the basic sciences as applied to ophthalmology, to be given on Saturday mornings, from 8:00 a.m. to 12:00 noon. These are designed for the resident staff in ophthalmology in preparation for Board examinations. All physicians, however, are cordially invited to attend the entire course or the individual lectures they desire. There are no fees for attendance. The lectures will be held in the Glaucoma Clinic (Room GA-E-38) on the ground floor of the Washington Hospital Center.

The faculty includes: Dr. Paul Boeder, State University of Iowa; Dr. Paul Calibrisi, George Washington University; Dr. David D. Donaldson, Harvard Medical School; Dr. Harry Green, Smith, Kline & French Laboratories, Philadelphia; Dr. Seymour P. Halbert, Columbia University; Dr. Irving H. Leopold, University of Pennsylvania; Dr. Edward W. D. Norton, University of Miami; Dr. Arthur M. Silberstein, Armed Forces Institute of Pathology; Dr. George K. Smelser, Columbia University; Dr. Carl J. Witkop, National Institute of Dental Research; and Dr. Lorenz E. Zimmerman, Armed Forces Institute of Pathology. The Ford Fund Lecturer in Residence, Dr. Edward W. D. Norton, will also give clinics and rounds for two days previous to his scheduled lectures. Dr. Boeder will also give three evening lectures in addition to his scheduled lectures. Further announcements of these addenda will be made during the course.

For the schedule of the basic science lectures and for further information write:

The Training and Education Committee
Department of Ophthalmology
Washington Hospital Center
110 Irving Street
Washington 10, D.C.

Gill Hospital meeting

The Gill Memorial Eye, Ear, and Throat Hospital, Roanoke, Virginia, will hold its 32nd annual spring congress in ophthalmology and otolaryngology and allied specialties, April 6 through April 11, 1959. Among the guest speakers invited to attend are: Joseph P. Atkins, Philadelphia; Walter S. Atkinson, Watertown, New York; Kenneth D. Bailey, Fairmont, West Virginia; William L. Benedict, Rochester, Minnesota; F. Willson Daily, Roanoke; Windsor S. Davies, Detroit; Jeff Davis, New York; Francis P. Furgiuele, Philadelphia; Surgeon General B. W. Hogan, U.S.N., Washington, D.C.; Bayard T. Horton, Rochester, Minnesota; Wendell L. Hughes, Hempstead, New York; John H. King, Washington, D.C.; T. Keith Lyle, London; George T. Nager, Baltimore; Robin M. Rankow, New York; Kenneth L. Roper, Chicago; A. D. Ruedemann, Detroit; Russell A. Sage, Indianapolis; Jules G. Waltner, New York; Edgar N. Weaver, Roanoke; Walter L. Winkenwerder, Baltimore.

Industrial eye problems

A four-day course in eye care and industrial eye programs will be presented by the Institute of Industrial Health and the Department of Ophthal-

mology of the University of Cincinnati on March 9 through 12, 1959. The course will be open to physicians only with preference given to men in active industrial practice. The objective of the course is to furnish the industrial physician with up-to-date information regarding eye care in industry.

The course will include basic principles of ophthalmology as well as the practical aspect of an industrial eye program.

In addition to the members of the faculty of the University of Cincinnati, the guest faculty will include persons well known in the field of ophthalmology and industrial ophthalmology.

For additional information and application write:
Secretary
Institute of Industrial Health
Kettering Laboratory
Eden and Bethesda Avenue
Cincinnati 19, Ohio.

Wills clinical conference

The 11th annual clinical conference of the Wills Eye Hospital staff and Society of Ex-Residents will be held in Philadelphia at the hospital on Thursday, Friday and Saturday, February 19, 20, and 21, 1959. A number of new and attractive features are planned by the committee for this event.

The Arthur J. Bedell Lecture will be delivered by Dr. Brittain F. Payne on "Complications following cataract surgery." In addition to individual presentations and color television surgery, clinical, surgical, and research subjects, the scientific program will include special surgical clinics on Thursday.

On Thursday evening, February 19th, there will be a scientific meeting of the Section on Ophthalmology of the Philadelphia College of Physicians which will be preceded by dinner. Everyone is invited to attend. Dr. R. N. Berke will be the guest speaker on the subject of "Surgical treatment of blepharoptosis."

The social activities will include an informal reception for all those who attend on Friday evening, February 20, 1959. The conference will be concluded by a dinner meeting of the Society of Ex-Residents of the hospital.

Miscellaneous

Stanford University postgraduate conference

The Stanford University School of Medicine, San Francisco, presented a four-day postgraduate course, November 12th through 15th. Topics covered were "Causes of failure in retinal detachment," "Light coagulation therapy for retinal detachment," "Causes of failure in glaucoma surgery," "Primary and secondary glaucoma complications and demonstrations of applanation tonotomy and tonography," "Best types of cataract sutures and wound healing," "Recent advances in drug therapy," "Complications of systemic diseases," and "Pigmented tumors of the uveal tract." On November 14th, a special lecture was given by Dr. G. Meyer-Schwickerath on "Light coagulation therapy of the eye."

The faculty of the conference included Drs. Jerome W. Bettman, Gilbert W. Cleasby, Baynard H. Colyear, Jr., William J. Ferguson, Jr., Milton Flocks, M. Wallace Friedman, Rufus C. Goodwin, Earle H. McBain, Dohrmann K. Pischel, Thomas C. Stevenson, Edward Tamler, Frank C. Winter, and Harold C. Zweng.

Societies

District of Columbia officers

The officers of the Section on Ophthalmology, The Medical Society of the District of Columbia, Washington, for the 1958-59 sessions are: President, Dr. Marshall M. Parks; vice-president and program chairman, Dr. Walter J. Romejko; secretary-treasurer, Dr. Elbert W. Dodd, Jr. The remaining two of the four scheduled meetings will be on February 10 and April 16, 1959.

Montreal meeting

The first quarterly meeting of the Montreal Ophthalmological Society for the year 1958-59, Dr. Roland Cloutier, presiding, was held at the Royal Victoria Hospital, October 30th. The guest speaker was Dr. Walter S. Atkinson of Watertown, New York, whose subject was "Measures which decrease complications of cataract extraction." The following papers were also presented: "Keratoplasty in Fuchs' dystrophy," Dr. G. Swartz; "Problems in neuro-ophthalmic diagnosis," Dr. S. B. Murphy; "The collection, storage, and selection of human vitreous for use in retinal detachment surgery," Dr. K. G. Edwards; "Office management of the patient with a reading difficulty," Dr. J. V. V. Nicholls.

Personal

Dr. Ralph W. Ryan, Morgantown, Pennsylvania, was a guest speaker at the Conference on Industrial Ophthalmology, held in Johannesburg, Union of South Africa, November 10th through 12th. Dr. Ryan also presented a paper on "Toxoplasmosis as related to eye disease," before the annual meeting of the South African Ophthalmological Society in Johannesburg.

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SUBJECT INDEX

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INDEX

Authors and Titles

of

Original Articles and Notes, Cases, Instruments

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